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CONGENITAL PULMONARY ARTERIOVENOUS ANEURYSM*

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I. INTRODUCTION

DURING the past decade clinical interest in congenital vascular malformations has increased markedly, the main impetus being provided by the development of surgical procedures to correct or relieve many of these anomalies. Among those malformations receiving increased recognition is one involving the pulmonary vascular bed, an anomaly basically hemangiomatous in nature. This has been described under a variety of names in the literature, including pulmonary arteriovenous aneurysm, pulmonary arteriovenous fistula, hemangioma or cavernous angioma of the lung, congenital telangiectasis of the lung and hemorrhagic telangiectasia with pulmonary artery aneurysm. While some of these terms have definite merit, congenital pulmonary arteriovenous aneurysm has been selected because of its wide usage and general acceptance. A review of the literature reveals well over eighty articles dealing with the subject, these publications ranging from short case reports to fairly comprehensive reviews. While there has been a certain amount of duplication in the reporting of individual cases, a reasonable search has revealed 85 apparently different reported examples of this condition. Of these 63 have been proved by surgery or autopsy, and in the remaining 22 unproved cases the diagnosis seems reasonably certain. Wilkens[86] is generally given credit for having been the first to describe the anomaly as seen at autopsy, in a case reported in 1917. It was not until 1937, however, that the diagnosis was first made during life,[82] and the first successful surgical attack took place in 1940.[46]

The incidence of pulmonary arteriovenous aneurysms is difficult to evaluate accurately, in view of the considerable number of cases reported during the past decade and their rarity in the literature prior to that time. It seems obvious, however, that the anomaly is relatively uncommon. In 15,000 consecutive autopsies at The Johns Hopkins Hospital only 3 such lesions were detected. Two of these were quite small, being incidental findings at autopsy. The remaining case was of clinical significance, and has been the subject of a previous case report.[81] It is doubted that this represents the true incidence of the condition, since small vascular anomalies of this type in the

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lung could be so easily overlooked in routine autopsy studies. There is apparently no significant sex preference, there being 45 males and 37 females in the 85 reported cases, with the sex not being stated in 3 instances. In many of the reported cases the race has not been recorded, but on only 2 occasions has it been stated that the patients were of the Negro race.

Frequently pulmonary arteriovenous aneurysms have been but one manifestation of a generalized angiomatous tendency. In 39 instances hemangiomas were reported elsewhere in the body, the majority of these being of the telangiectatic type. Attention has been called to the increased incidence of pulmonary arteriovenous aneurysms in patients with hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease), and in 14 of the 85 reported cases there has been a fairly clear family history of this disease. On 2 occasions siblings have been found to have pulmonary arteriovenous aneurysms.[39,61]

It is our intention at this time to discuss the pathological anatomy, disturbed physiology and clinical aspects of congenital arteriovenous aneurysms of the lung, and to present 9 cases of the malformation which have been seen at The Johns Hopkins Hospital.

II. PATHOLOGICAL ANATOMY

Pathologically, arteriovenous aneurysms of the lung are similar to hemangiomatous malformations found elsewhere in the body. They are composed of two basic elements: (1) vascular channels lined with endothelium; and (2) a supporting connective tissue stroma. The vascular channels range from capillary sized structures to huge tubular or saccular multilobulated dilatations with walls of varying thickness, and there may be considerable variation in the amount of associated connective tissue elements. Capillary hemangiomas within the lung, composed of small vessels, have usually been incidental findings at autopsy as have been the pinpoint sized angiomas or telangiectases occasionally encountered. Most of the clinically significant arteriovenous aneurysms of the lung are fundamentally hemangiomas of the so-called cavernous type, being composed of large tortuous and irregular channels with relatively thin walls and scant connective tissue stroma. Usually one border of these lesions presents on the pleural surface of the lung, and it apparently has been an impressive sight at surgery to see these bulging and pulsating vascular sacs with walls frequently so thin that the blood can actually be visualized swirling and streaming within their lumens. In view of the thinness of the walls, it is not surprising that occasionally rupture may occur into the pleural space or into the tracheobronchial tree. Small calcified plaques have been encountered within pulmonary arteriovenous aneurysms,[7,23,50] but apparently phlebolith-like calcifications have not been noted.

The vascular supply and pattern of these arteriovenous aneurysms is of considerable importance. Unfortunately, routine pathological techniques are not adequate for an accurate understanding of the complexities that may occur in the vascular channels and communications within these lesions. As a result, many of the proved cases have not been ideally studied. The most satisfactory method for demonstrating the vascular anatomy has been to inject materials into the vessels supplying the area and to obtain an actual cast of the lumen of the channels within and around the anomaly.[54] Surgical and pathological studies have revealed that the major arterial supply is usually from a branch or branches of the pulmonary artery, and the venous drainage empties into the pulmonary vein system. Frequently, the arterial and venous branches supplying the aneurysm may be considerably dilated, and this dilatation may extend for a variable distance towards the heart. Not only may these supplying trunks be dilated, but their walls may also be considerably thinner than normal. This phenomenon has been encountered in large arteriovenous aneurysms elsewhere in the body, as well as in traumatic arteriovenous

fistulas involving the systemic circulation. In most of the clinically significant pulmonary arteriovenous aneurysms which have been carefully studied there have been large channels providing more or less direct communication between the afferent arterial supply and the efferent venous drainage. This shunt in parallel, as it were, permits varying amounts of pulmonary arterial blood to be short circuited into the pulmonary vein. Since this blood bypasses the pulmonary capillary bed, varying amounts of unoxygenated blood are returned to the left side of the heart. Some authors have suggested that the bronchial arterial system may enter into the composition of the vascular supply to pulmonary arteriovenous aneurysms.[7,51] Rarely, there may be anomalous arterial or venous trunks supplying these lesions,[6,50,54] one of the most striking being the case reported by Watson[81] in which the major arterial supply to an arteriovenous aneurysm of the right lower lobe seemed to be from an aberrant artery arising from the lower thoracic aorta. When this vessel was compressed at surgery, the aneurysm no longer pulsated. Occasionally at surgery the arteriovenous aneurysm or adjacent lung has been found to be attached to the parietal pleura and chest wall by unusually vascular adhesions,[2,7,39] suggesting the establishment of a secondary vascular supply.

Anatomically, pulmonary arteriovenous aneurysms may be found anywhere in the lung. The location by lung and lobes in the reported cases is tabulated in Table 1.

The discrepancy between the number of arteriovenous aneurysms found and the number of reported cases stresses the frequency with which there may be multiple lesions in the same individual. Twenty-seven patients had multiple pulmonary arteriovenous aneurysms, with involvement of two or more lobes. In 21 instances lesions were present in both lungs, and in one case the presence of aneurysms in all five lobes was surgically confirmed.[1,2] Also of importance is the fact that a careful search at surgery or autopsy has frequently revealed additional lesions whose presence had not been detected by clinical means. These have varied from small pinpoint angiomas to lesions of moderate size.

Table I

LOCATION OF PULMONARY ARTERIOVENOUS ANEURYSMS IN 85 REPORTED CASES*

Right Lung		Left Lung	
Right upper lobe	12	Left upper lobe	18
Right middle lobe	17	Left lower lobe	20
Right lower lobe	32	Unstated	12
Unstated	4		
	—	Total	50
Total	65		

* In 3 instances there was no mention of the localization of the aneurysms, either as regards lung or lobe involved.

Pulmonary arteriovenous aneurysms do not seem to cause any significant disturbance in the adjacent lung parenchyma. There is occasionally some minimal compression of the surrounding lung, but there have been no reports of associated atelectasis or bronchiectasis.

III. PHYSIOLOGICAL ASPECTS

Physiological disturbances associated with pulmonary arteriovenous aneurysms may be totally absent or quite marked, depending upon the amount of unoxygenated blood which is shunted from the pulmonary artery through the aneurysm into the systemic circulation. Relatively few of the reported cases have been carefully studied from a physiological aspect,[7,55] and most of those which have been so studied have had obvious clinical evidence of a significant shunt. In 6 cases the percentage shunt has been estimated; that is, the per cent of the right ventricular output which passes through the shunt and bypasses the pulmonary capillary bed. The recorded estimates have ranged from 18[79] to 89[52] per cent, the intermediate figures being 28, 45, 58 and 80 per cent. When significant amounts of unoxygenated blood are returned to the left side of the heart, varying degrees of oxygen unsaturation occur in

the systemic circulation. The obvious clinical finding resulting from this unsaturation is cyanosis of varying intensity. But more important is the influence of the resulting anoxemia upon the patient as a whole and upon the hemopoietic system specifically. There is a stimulation of the erthropoietic elements of the marrow, with a resultant polycythemic response. This results in a variable elevation of the red blood cell count and hemoglobin, the maximum elevation of the red count reported being 11.4 million.[40] There is also an elevation of the total blood volume due to the increased red cell mass, there being no significant elevation of the plasma volume or increase in the white blood cell count. The response of the hemopoietic system in such cases is similar to that produced by the anoxemia of high altitudes.[47]

It is of some interest to speculate upon the combination of size and number of arteriovenous aneurysms necessary to produce a clinically significant shunt. Almost invariably in the reported cases there have been one or more large aneurysms present. Theoretically, however, it is conceivable that a large number of very small lesions could produce the same result. The summation of the minute shunts through a large number of telangiectatic angiomas might approach that occurring in one or more larger lesions. There is one reported case suggesting that this may occur,[18] although the presence of multiple pulmonary telangiectases had not been positively confirmed at the time of the report.

It is usually stated that there is no significant disturbance in the over-all circulatory dynamics in cases of pulmonary arteriovenous aneurysms. The blood pressure, pulse rate and circulation time are typically unaltered. In those few cases which have been catheterized, the right atrial, ventricular and pulmonary artery pressures have been essentially normal, and the cardiac output within normal limits. These findings in pulmonary arteriovenous aneurysms are in distinct contrast to congenital or acquired lesions in the systemic circulation which permit the shunting of significant amounts of arterial blood into the venous circulation. It is well recognized that profound disturbances in the cardiovascular dynamics may occur in these cases, with an elevation in the pulse pressure, pulse rate and cardiac output resulting in an increased workload on the heart and the danger of eventual cardiac failure. The usual explanation for the absence of such changes in lesions involving the pulmonary circulation is that the pulmonary artery pressure is normally so low that the presence of a shunt in parallel does not significantly reduce the over-all vascular resistance of the lung.[35]

While it is apparently true that the average pulmonary arteriovenous aneurysm does not cause any significant disturbance in circulatory dynamics, there is some suggestive evidence that this is not always the case. For example, 17 patients were noted to have cardiac enlargement either clinically or at autopsy. In 9 of these, the enlargement could be easily explained on the basis of other factors, such as hypertension or valvular heart disease. In 8 instances, however, the exact explanation was not obvious, although there was no specific pattern to the enlargement, either clinically or in the 4 cases in this group in which the heart was studied at autopsy.[52,68,86,88] In 7 adult patients with pulmonary arteriovenous aneurysm it was noted that there was a right axis deviation on the electrocardiogram, suggesting some right ventricular strain, and occasional patients have had episodes of unexplained cardiac failure. For these reasons, it is felt that the effect of pulmonary arteriovenous aneurysms on the circulatory dynamics may not always be as benign as is usually stated.

Relatively little experimental work has been done on the physiological disturbances associated with artificially produced pulmonary arteriovenous shunts.[15,60] The most thorough experimental study reported to date has been by Takaro *et al.*[83] These workers were able to create shunts in dogs

causing changes similar to those found in patients clinically.

It should be emphasized that not all pulmonary arteriovenous aneurysms produce a significant degree of arteriovenous shunt or cause obvious physiological disturbances. Most of the reported cases have had significant shunts, but this may be explained by the fact that such lesions tend to make themselves clinically apparent. In 3 of the 85 reported cases which were studied relatively carefully there were no obvious physiological disturbances,[26,54,70] indicating the absence of significant shunt, and in other instances the changes were minimal. These do not include cases in which complicating factors, such as anemia secondary to hemorrhage, tended to mask clinical evidence of a physiological response.

IV. CLINICAL ASPECTS

The clinical aspects of pulmonary arteriovenous aneurysms, based on an analysis of previously reported cases, will be discussed under the headings of symptomatology, physical findings, laboratory data, roentgenological findings, treatment and outcome.

1. *Symptomatology.* A reasonable amount of clinical information was recorded in 79 of the 85 reported cases. Of these, 71 had symptoms of some type, while 8 patients were said to be totally asymptomatic. In Table II the symptoms are grouped according to their frequency.

Dyspnea, resulting from anoxemia, was by far the most common symptom. There was considerable variation in its intensity, but more often than not it was exertional in type and relatively mild. Epistaxis is not regarded as a symptom of pulmonary arteriovenous aneurysm per se, but rather as a manifestation of telangiectases in the mucosa of the nasal airways. A similar explanation accounts for the occasional visceral hemorrhages noted. Neurological disturbances were relatively frequent. These were usually transitory in type, ranging in intensity from slight sensory or motor disturbances to grand mal convulsive episodes. Occasionally permanent damage, such as hemiplegia, has been noted. It is presumed that these episodes represent cerebral insults of varying intensity due to anoxemia or thrombosis and that polycythemia plays an important role,[13] although few of these cases have had autopsy confirmation. Occasionally, it has been suggested that these episodes are the result of air emboli, the air gaining entrance to the circulatory system through a defect in the wall of the aneurysm,[53] but this view has not gained general acceptance. The hemoptyses varied from repeated minor episodes over a period of years to a single massive fatal hemorrhage. It occasionally followed physical exertion, and it has usually been thought that the blood came from a leak in the aneurysm. It should be remembered, however, that telangiectases in the tracheobronchial tree can also bleed and cause hemoptysis. The chest pain or discomfort was frequently substernal in location, and usually not of a severe nature.

In many instances the degree of subjective incapacity resulting from the arteriovenous aneurysm was quite minimal. Many of these patients were able to carry on normal activity quite well, noticing limitations only upon physical exertion. Sixty-three of the patients were between the ages of twenty-one and seventy years at the time their case was reported, and in most of these, the clinical history dated back many years. Often there was no significant increase in the degree of symptomatology, and these facts tend to sub-

Table II

SYMPTOMATOLOGY RECORDED IN 79 CASES OF PULMONARY ARTERIOVENOUS ANEURYSM

Symptoms	No. Cases
Dyspnea	44
Epistaxis	26
Cerebral disturbance	22
Hemoptysis	17
Chest pain	12
None	8

stantiate the concept that pulmonary arteriovenous aneurysms may have a prolonged and relatively benign clinical course.

2. *Physical Findings.* A summary of the pertinent findings on physical examination is presented in Table III.

Table III

PERTINENT PHYSICAL FINDINGS IN 85 REPORTED CASES OF PULMONARY ARTERIOVENOUS ANEURYSMS

Finding	Present	Absent	Not Recorded
Pulmonary murmur	52	21	12
Cyanosis	61	13	11
Clubbing	59	13	13
Hemangiomas of skin or mucous membrane	38	12	35

Abnormal murmurs were detected over the region of the aneurysms in 52 instances. These varied considerably in intensity and quality in individual cases, but were usually either continuous in nature with a systolic accentuation or were heard only during systole. Frequently, the murmurs were noted to increase in intensity during or at the end of deep inspiration. Only rarely could a thrill be palpated.[21,32,65] Cyanosis and clubbing were frequent findings. Associated telangiectases or hemangiomas in the skin or mucous membranes were noted on routine physical examination in 38 instances.

There were no other consistent physical findings. The patients were usually reasonably well developed and nourished, and save for the murmur over the arteriovenous aneurysm the heart and lungs were usually unremarkable. The absence of cardiac murmurs would tend to exclude congenital cardiac malformations as a cause of cyanosis. As noted previously, the blood pressure, pulse and respiratory rates were usually within normal limits. The spleen was only rarely enlarged, helping to differentiate this condition from polycythemia vera.

3. *Laboratory Data.* Of the various routine hematologic studies, the red blood cell count, hematocrit and hemoglobin are the only ones to show significant alteration with any frequency. In 71 instances the red blood cell count was recorded. There was significant elevation of the count in 60 cases, but a normal or anemic count was noted in the remaining 11 patients. In several of the patients with subnormal counts, there was a history of epistaxis or other hemorrhage accounting for the anemia. With control of the hemorrhage and supportive therapy an elevation of the red blood cell count from anemic to polycythemic levels has been noted.[5] With the elevated red blood cell counts there was usually a proportional elevation of the hematocrit and hemoglobin levels. The white blood cell and differential counts as well as that of the platelets showed no significant alterations and helped to exclude polycythemia vera from the list of differential diagnoses. The systemic arterial oxygen saturation was recorded in 30 cases. In only 1 instance was the figure found to be within normal limits,[54] but in general the examination was only performed in those patients with clinically obvious cyanosis. The recorded red blood cell counts and systemic arterial oxygen saturations are summarized in Table IV.

The electrocardiogram was usually stated to be normal, although as noted ear-

Table IV

RECORDED RED BLOOD CELL COUNT AND SYSTEMIC ARTERIAL OXYGEN SATURATION IN CASES OF PULMONARY ARTERIOVENOUS ANEURYSMS

Red Blood Cell Count		Systemic Arterial Oxygen Saturation	
Million	No. Cases	Per Cent	No. Cases
2–5	11	51–60	2
5–8	50	61–70	5
8–11	10	71–80	9
Not recorded	14	81–90	13
	—	91–96	1
Total	85	Not recorded	55
			—
		Total	85

lier several instances of right axis deviation were recorded. No consistent changes in the circulation time were recorded.

4. *Roentgenological Findings.* In all of the reported pulmonary arteriovenous aneurysms in which roentgenograms of the chest were taken some abnormality was detected within the lung fields. The typical lesion, on routine roentgenography, is a rounded or lobulated homogeneous density within the lung parenchyma having relatively well defined borders and being connected to the hilar region by cord-like bands. It has been suggested that this pattern is quite typical and specific, but it seems fair to say that a high index of suspicion is of considerable help in making the roentgenologic diagnosis. Not all of the lesions have been so typical in appearance, and it is not surprising that a diagnosis of bronchiectasis, tuberculosis and primary or metastatic neoplasm has been entertained on occasions. Calcification within the aneurysm is uncommon, and when seen has been plaque-like in appearance.[7,26,50] Roentgenoscopic study should be performed routinely. Intrinsic pulsations were noted in 18 instances, and were said to be absent in 12 cases. Not infrequently unusually active pulsations were observed within hilar vessels, and pulsations within the communicating vessels in the lung parenchyma may be seen. Several authors have commented upon the fact that the diameter of the aneurysm may increase with a reduction of intrathoracic pressure (Müller maneuver) and decrease with a rise in intrathoracic pressure (Valsalva experiment). Roentgen kymography may confirm some of these findings. Laminagraphy frequently helps to establish the nature of the lesion, the tortuous vessels in and around the anomaly often standing out with surprising distinctness.

By far the most definitive roentgenologic procedure is angiocardiography. This has been employed in 30 cases, and correctly showed the nature of the process in 26 instances. In 3 of the remaining cases the examination was considered to be technically unsatisfactory. In the final case it was thought clinically that the patient had multiple telangiectatic angiomas throughout the lungs,[18] and in this instance angiocardiography apparently demonstrated no abnormalities. The large arterial and venous trunks supplying the anomaly are usually well opacified, as are the tortuous channels and sacculations within the arteriovenous aneurysm. These vessels usually reach their maximum density within two to three seconds following filling of the pulmonary artery. With large arteriovenous shunts it has occasionally been noted that the vascular tree elsewhere in the lung does not opacify normally, and that the opaque material may appear in the left atrium with abnormal rapidity.[4] There has been one reported fatality following angiocardiography,[81] with the patient dying of pulmonary edema twenty-five minutes following the injection of the medium. This patient's clinical picture was somewhat complicated, however, and there had been previous episodes of pulmonary edema. Another patient developed a large hemoptysis and apnea lasting two or three minutes in conjunction with angiocardiography.[74] In both instances the contrast substance was introduced via catheters, the tip being placed in the right atrium in the fatal case and in the main pulmonary artery in the nonfatal reaction.

The minimal size of the arteriovenous aneurysm or its vascular channels necessary for angiocardiographic demonstration has not been established, and it is probable that telangiectases and other small angiomas would be overlooked. Nevertheless, it would seem that angiocardiographic study is well justified in any case in which surgical interference is being considered, in an effort to clearly establish the extent and number of arteriovenous aneurysms present.

5. *Treatment.* Three forms of specific therapy have been tried in the treatment of pulmonary hemangiomas, two of these being of only historical interest. Pneumothorax has been attempted in the hope that the vascular channels within the aneurysm

would collapse along with the lung, but in the few cases tried this did not prove successful.[46,50] Roentgen therapy has also been utilized occasionally but without obvious benefit.[50,69] Surgery appears to offer the only logical method of definitive therapy, and this has been employed in 44 of the 85 reported cases. Removal of the lesion was undertaken in 41 instances, most commonly by lobectomy and less frequently by segmental resection or total pneumonectomy. In 3 instances the feeder vessels supplying the arteriovenous aneurysm were ligated,[4,63,84] the aneurysm being left in place. Four patients with multiple arteriovenous aneurysms involving both lungs have had bilateral thorocotomies, one of these having a total of six aneurysms removed in a two-stage operation.[1,2] When bilateral lesions have been present, it has usually been elected to remove the largest lesion first. Two such patients are of interest[1,6] in that there seemed to be clear evidence that the remaining arteriovenous aneurysms tended to enlarge in the intervals between surgery, and initial clinical improvement and evidence of a decrease in the amount of shunt were only temporary.

There have been 4 deaths in the immediate postoperative period among the 44 patients coming to surgery.

The indications for surgical interference have not been completely established. All agree that surgery is justified in the patient with an isolated lesion causing hemoptysis or significant symptoms secondary to anoxemia and polycythemia, or even in symptomatic cases with a limited number of lesions involving one or both lungs. The most difficult decisions occur in those patients with isolated lesions in which symptoms are minimal or absent and in which there is no clinical evidence of a marked shunt, or in those patients with extensive and multiple lesions where successful therapy would necessitate the removal of large segments of pulmonary tissue in several stages.

6. *Outcome.* In discussing the natural course of the disease and its outcome, the 85 reported cases have been divided into two groups, depending on whether or not surgical therapy was attempted. In 41 instances the patients did not receive surgery, and their status at the time of their report in the literature is summarized in Table v.

Table V

REPORTED STATUS OF 41 UNTREATED PULMONARY ARTERIOVENOUS ANEURYSMS

Status		No. Cases
Alive with absent or minimal symptoms		15
Alive with moderate to marked symptoms		5
Dead		21
Death related to pulmonary lesion	11	
Death not related to pulmonary lesion or cause unstated	10	
Total		41

Twenty of these patients were alive at the time their cases were reported, and the majority of these were having relatively minor symptoms. Many of them had been followed for a considerable length of time, often with relatively little progression in the degree of symptomatology. Twenty-one patients were dead. In 10 of these death was either not related to the pulmonary arteriovenous aneurysm or its cause was not obvious. These include those instances in which the aneurysms were incidental findings at autopsy. In 11 patients, however, death did seem to be related to the pulmonary lesion. Three patients died as a result of rupture of the aneurysm into the pleural space, and in 2 instances death was secondary to massive hemoptysis. There were 3 proved brain abscesses, and in another patient death was secondary to an undetermined intracranial catastrophe. An increased incidence of brain abscesses has been noted in other congenital vascular anomalies, and probably the same etiological factors play a role in those instances where pulmonary arteriovenous aneurysms were present.[13] There was 1 fatality from

myocardial infarction, perhaps secondary to anoxemia and polycythemia, and 1 death following angiocardiography.

The data summarizing the outcome in the 44 surgically treated patients are presented in Table VI.

TABLE VI

REPORTED STATUS OF 44 SURGICALLY TREATED PULMONARY ARTERIOVENOUS ANEURYSMS

Status	No. Cases
Apparent clinical cure	23
Alive with evidence of disease	4
Postoperative pulmonary insufficiency	2
Dead	5
Unstated	10
Total	44

In 23 instances there was an apparent clinical cure following surgery, although careful and prolonged postoperative studies were frequently not available. Four patients appear to have remaining lesions elsewhere in the lungs. At least 2 patients were left with some pulmonary insufficiency secondary to the removal of large amounts of lung parenchyma. There were 4 operative deaths in the 5 fatalities, the other patient dying of an intracranial accident some time after surgery. In the remaining 10 cases little comment was made concerning the postoperative status.

V. REPORT OF CASES

Between 1940 and 1952 a total of 10 patients with clinically evident pulmonary arteriovenous aneurysms were seen at The Johns Hopkins Hospital. One of these cases has been the subject of a previous complete case report,[59] and will not be discussed here. Another patient, presented in the literature previously as an unproved example of multiple pulmonary arteriovenous aneurysms,[56] had surgical confirmation at our institution and will be included (Case v). Of the 9 cases to be reported, 7 have had surgical or autopsy confirmation and 2 are still unproved. Physiological studies, including catheterization, were performed by Dr. Richard Bing and his associates in 5 instances, and these data have been the subject of a previous report.[35]

CASE I. E. B. (J.H.H. 215050), a white female, aged fifty-two, was admitted for the first time on November 9, 1940, for evaluation of long-standing cyanosis and dyspnea. The family and past histories were noncontributory. The present illness was of many years' duration. Exertional dyspnea of gradually increasing intensity had been present for twenty years, and cyanosis had been noted for ten years. Six years prior to admission, a chest film is said to have revealed a lesion in the right lung. She gradually developed increasing fatigue and weakness, and following an attack of grippe eight months prior to admission, she became so weak that she could hardly walk across a room. There had been no epistaxes, hemoptyses or cerebral disturbance.

On physical examination the pulse was 92, respirations 20 and the blood pressure 128/32. She was well developed and nourished. Positive findings included cyanosis and a distinct bruit with systolic accentuation over the lower half of the right chest. There was no clubbing, nor were hemangiomas noted in the skin or mucous membranes. Laboratory studies revealed a white blood cell count of 5.7 million, hemoglobin 18.2 gm., hematocrit 58 and a white blood cell count of 7,200, with a normal differential count. The total blood volume was 121 per cent of normal, the increase being due entirely to the elevated red cell mass. The peripheral arterial oxygen saturation was 88.5 per cent. An electrocardiogram revealed no significant changes.

Routine chest films revealed a normal sized heart. There was a large area of mottled density at the right lung base with prominent strands extending into the right hilus (Fig. 1). No pulsations were detected at roentgenoscopy, and roentgenologically it was suspected that the process represented a pneumonitis. Clinically, however, the possibility of a pulmonary arteriovenous anastomosis of some type was considered.

The patient's marked subjective weakness was not easily explained. While there was no clear evidence of Addison's disease, she seemed to feel much better with percorten and salt, and because of this five pellets of desoxycorticosterone acetate were implanted subcutaneously and she was discharged on 4 grams of sodium chloride daily.

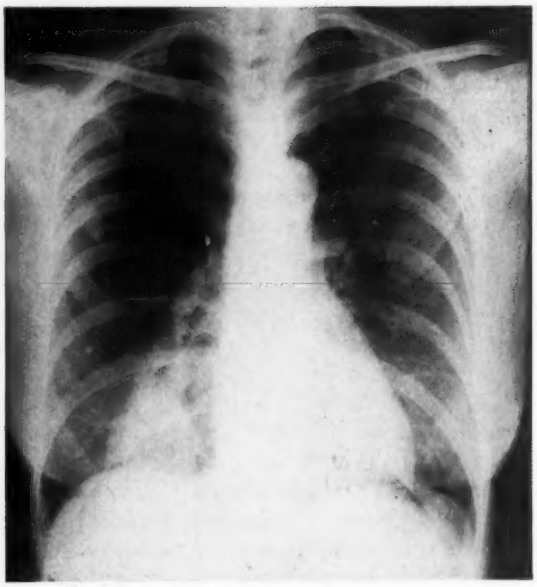

Fig. 1. Case 1. A proved arteriovenous aneurysm involving predominantly the right middle lobe. Roentgenographically the anomaly presents as a rounded density at the right base with prominent strands extending into the right hilus.

Death occurred six years later from a cerebral hemorrhage. During the last year of her life, she had a sustained hypertension which was presumed to be secondary to the desoxycorticosterone which she seemed to require up to the time of her death. At autopsy elsewhere* the left lung was normal. A typical arteriovenous aneurysm was found at the right base, apparently lying predominantly in the middle lobe and involving the interlobar fissure.

Comment. With the exception of the profound weakness and an appearance simulating Addison's disease, this case is typical of a pulmonary arteriovenous aneurysm with a significant arteriovenous shunt and slowly progressing symptomatology extending over a period of twenty-six years.

Case II. H. M. (J.H.H. 514620–A62954), a white female, aged twenty-eight, was first seen on May 24, 1948, with the chief complaint of cyanosis. The family and past histories were noncontributory. The present illness dated back to early childhood. She was thought to be normal at birth, but dyspnea and cyanosis were noted at the age of one and one-half years, at which time a diagnosis of congenital heart disease was made. At the age of seventeen years she had "rheumatic arthritis" and was in bed for a month. Her exercise tolerance had always been quite limited, and at the time of her first visit she was able to walk only a few blocks. There had been no epistaxes, hemoptyses or cerebral episodes.

On physical examination the pulse was 90, respirations 24 and the blood pressure 90/75. She was poorly developed and nourished, and there was marked cyanosis and clubbing. A few small cutaneous angiomas were noted. The heart and lungs were clear, without pulmonary murmurs. Laboratory examinations revealed a red blood cell count of 8.9 million, hemoglobin 25 grams, hematocrit 77 and a white blood cell count of 8,000. The femoral arterial oxygen saturation was 62 per cent. The circulation time, arm-to-tongue with decholin, was nine seconds, and an electrocardiogram showed no distinctive abnormalities.

Routine chest films and roentgenoscopy revealed an essentially normal heart. The main trunk of the pulmonary artery seemed enlarged, as did its hilar branches on the right, and active pulsations were noted in these areas. Mottling and prominence of the vascular markings were present in the region of the right lower lobe (Fig. 2*A*). Roentgenologically it was felt that there was some abnormality within the pulmonary vascular pattern in this area, but the correct diagnosis was not suggested. Clinically it was felt that she had one of the more complicated malformations of the heart, perhaps with some associated anomaly within the lung.

The patient was next seen sixteen months later, at which time there had been little change in the symptomatology and clinical findings. On this visit she was catheterized, at which time the correct diagnosis was made. The right ventricular and pulmonary artery pressures were thought to be minimally elevated, and the cardiac output was slightly above normal. There was no evidence of an intracardiac abnormality. It was estimated that 76 per cent of the right ventricular output was shunted through the lesion in the lung. Laminagrams of the lower two-thirds of the right hemithorax revealed numerous dilated and tortuous vascular channels radiating outward and downward from the region of the right hilus. The majority

* Dr. W. H. Willis of the Slocum-Dickson Clinic in Utica, New York, was kind enough to supply us with the autopsy findings.

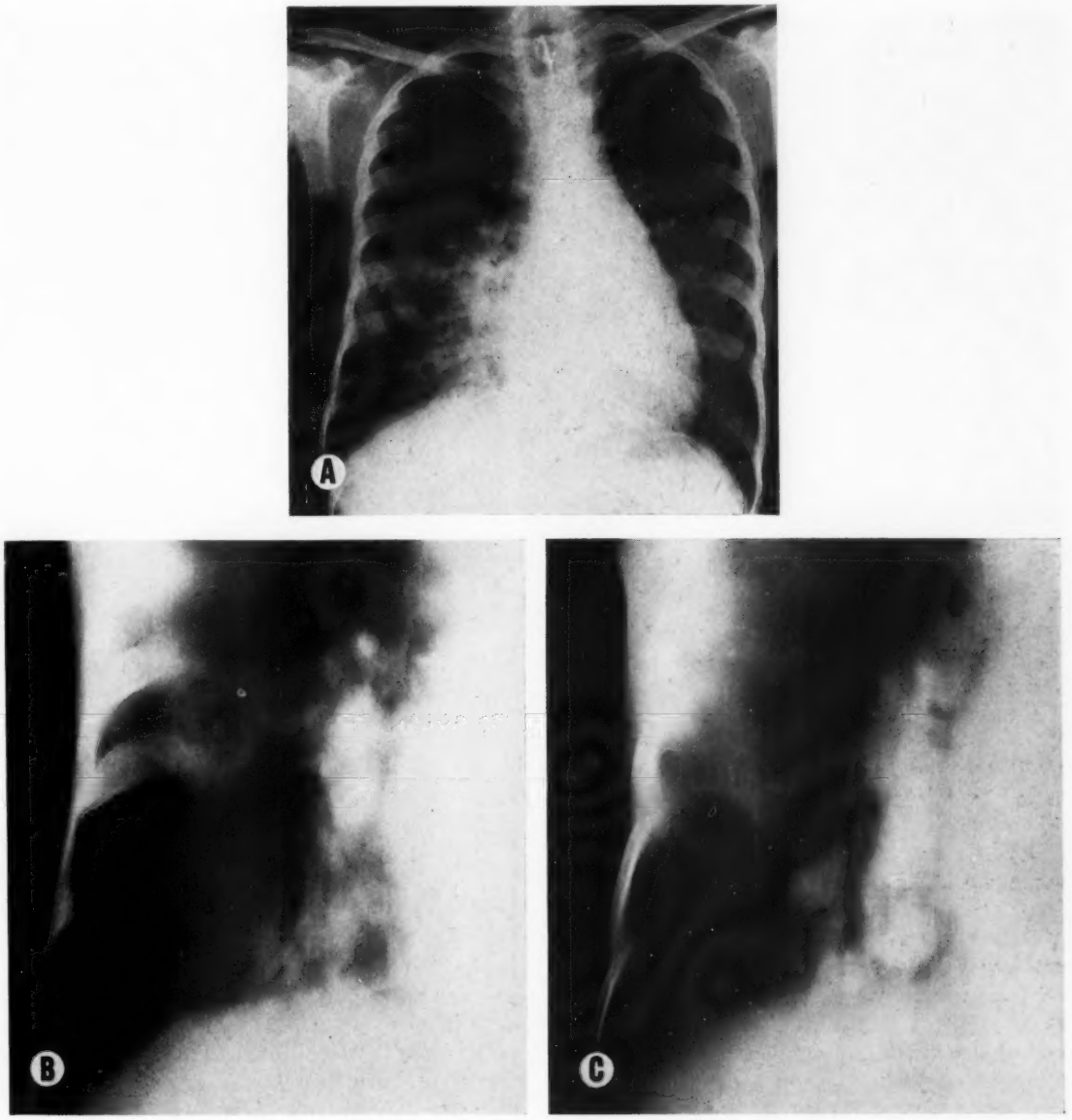

Fig. 2. Case 11. (*A*) Proved arteriovenous aneurysms involving the right lower lobe. The mottling and prominence of the parenchymal vascular markings extending downward along the right heart border are clearly shown. (*B*) and (*C*) Representative laminagrams at different levels through the right lower hemithorax demonstrate the large vascular trunks communicating with the hilus, as well as the multiple, varying sized vessels within the aneurysms themselves.

of these were seen to lie medially and posteriorly in the region of the lower lobe (Fig. 2*B* and *C*). The roentgenologic impression was that there was a large congenital arteriovenous vascular tumor of the right lung, and that this lay predominantly if not entirely in the lower lobe.

A right thoracotomy was performed on September 23, 1949, by Dr. Alfred Blalock. Several typical arteriovenous aneurysms were found in the lower lobe, with tortuous vessels of all sizes being noted within the anomalies. The middle and upper lobes appeared to be normal, and dilated bronchial arteries were commented upon. A right lower lobectomy was performed. Her postoperative course was smooth, and on the sixth postoperative day the systemic arterial oxygen saturation was 96 per cent.

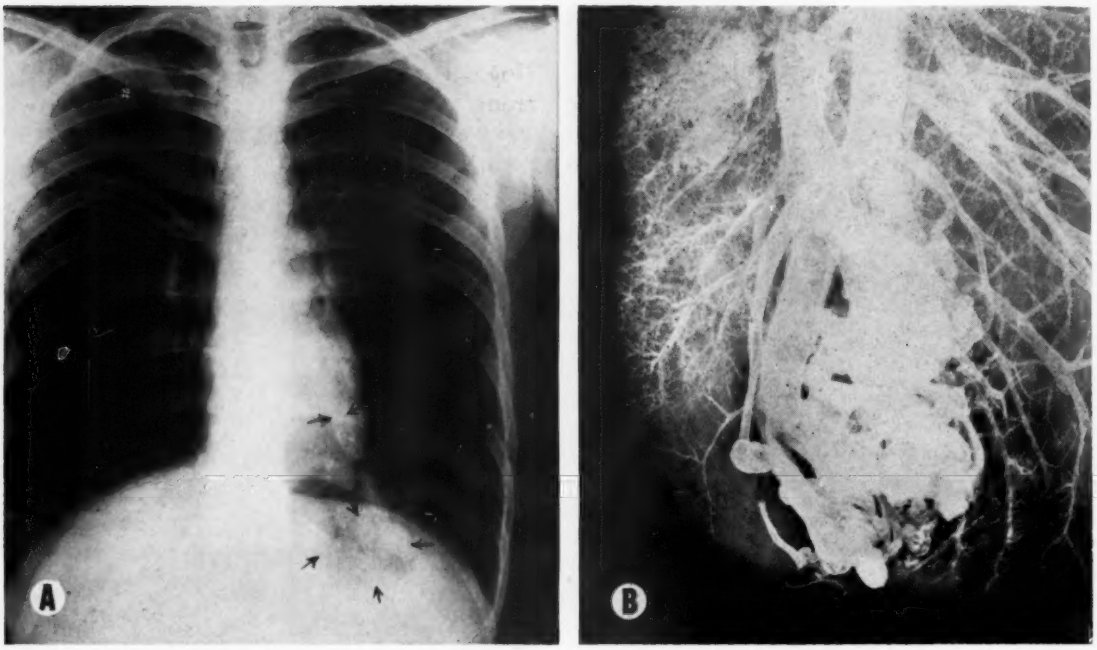

Fig. 3. Case III. (*A*) A proved arteriovenous aneurysm involving the left lower lobe. There are several rounded densities at the left base, which in the posteroanterior projection are partially visualized through the gas bubble in the stomach. Prominent vessels extend downward from the left hilus into the parenchymal densities. (*B*) Roentgenogram of the resected left lower lobe, following the injection of a barium suspension into the prominent arterial trunk supplying the arteriovenous aneurysm. The tortuous channels within the aneurysm are clearly shown, as is the large venous trunk draining the area.

Eight months postoperatively she returned for a check. At that time she was dramatically improved over her preoperative status. She was totally asymptomatic, and there had been a marked increase in her exercise tolerance. There was no visible cyanosis, although some clubbing of the fingers remained. Hematologic studies revealed a red blood cell count of 5.2 million, hemoglobin 12.5 gm., and a hematocrit of 43. A femoral arterial oxygen study was reported as 82 per cent, but the accuracy of this was questioned. A letter two and one-half years following surgery stated that she was leading a normal and active life, and that the clubbing had almost completely disappeared.

Comment. This is another example of symptomatic arteriovenous aneurysms limited to the right lower lobe, with apparent clinical cure following lobectomy.

Case III. H.H.M. (J.H.H. 478746), a white male, aged twenty-one, was admitted on October 23, 1948, for consideration of surgery for a pulmonary arteriovenous aneurysm. His family and past histories were noncontributory. The present illness dated back to childhood. The patient stated that his complexion had been rather ruddy as long as he could remember, and his parents had noted that as a child he became blue on swimming. At the age of seventeen years, during a routine physical examination, cyanosis and the presence of polycythemia were detected. He was told by several doctors that his heart seemed to be normal. Four months prior to admission the red blood cell count was found to be 8 million, and a careful diagnostic workup elsewhere revealed the correct explanation for the cyanosis and polycythemia, and surgery was recommended. Although the patient felt that there had been some increase in the degree of cyanosis in recent years, he denied having any real symptoms. In particular, there had been no exertional dyspnea, and he felt that his exercise tolerance was not limited in any way. There had been no cerebral disturbances, hemoptyses, epistaxes or episodes of chest pain.

On physical examination the pulse was 76, respirations 20 and the blood pressure 110/70. He was moderately well developed and

nourished. There was definite but not marked cyanosis, as well as moderate clubbing of the fingers and toes. No telangiectases were detected in the skin or mucous membranes. The heart was not enlarged or otherwise abnormal. No pulmonary murmurs or other unusual findings were detected over the lung fields on routine examination. Laboratory studies revealed a red blood cell count of 7.9 million, hemoglobin 24 gm., and a hematocrit of 66. The white blood cell count was 7,000, with a normal differential count. The femoral arterial oxygen saturation was 84 per cent.

Routine chest films and roentgenoscopy revealed no abnormalities in the heart or great vessels. At the left lung base posteriorly there were several rounded soft tissue densities measuring 1 to 2 cm. in diameter, and in the posteroanterior projection these were partially visualized through the gas bubble in the stomach (Fig. 3*A*). Rather prominent band-like densities were noted to extend from the inferior portion of the left hilus down to the parenchymal lesions. At the time of roentgenoscopy no intrinsic pulsations were detected in these areas. Another small area of increased density was noted underlying the right second interspace anteriorly. It was felt that the appearance of the lesion in the posterior basilar segment of the left lower lobe was quite compatible with an arteriovenous aneurysm, and that the secondary lesion in the right upper lobe could represent either an infectious process or another small arteriovenous aneurysm. Following the roentgenographic localization of the lesion in the left lower lobe, careful auscultation of the overlying chest wall revealed a small area in which a soft continuous murmur with systolic accentuation could be heard.

On October 28, 1948, Dr. Alfred Blalock performed a left thoracotomy. There were no adhesions between the lung and chest wall. A distinct aneurysmal-like lesion was found in the posterior basilar portion of the left lower lobe which could be emptied by pressure only to promptly fill again. There was one small area in the inferior portion of the upper lobe which might have been a minute angioma, and this was occluded with a through-and-through mattress suture. It was elected to do a left lower lobectomy, and this was performed without difficulty. The arterial supply of the resected lobe was injected with an opaque substance, and a specimen film clearly showed the vascular channels with the aneurysm, as well as the enlarged vessels supplying the anomaly (Fig. 3*B*). The prompt filling of the venous trunk indicates the presence of good sized arteriovenous communications within the aneurysm.

The patient's postoperative course was reasonably smooth, and on the fourteenth postoperative day a femoral arterial oxygen saturation was 98 per cent. He was discharged shortly thereafter, and no long-term followup has been obtained.

Comment. This is an example of a proved isolated arteriovenous aneurysm involving the left lower lobe. While there was clinical evidence of an arteriovenous shunt through the lesion, there was no significant symptomatology.

Case iv. S. X. (J.H.H. 467587–A63444), a white male, aged fifteen, was seen for the first time on June 10, 1948, for evaluation of intense cyanosis and clubbing. The family and past histories were noncontributory. The present illness dated back to childhood. The patient was thought to be quite normal until the age of five years, when cyanosis was first noted during a hospital admission for a tonsil and adenoid operation. This gradually increased in intensity, and there was progressive exertional dyspnea. At the age of seven years, a hemangioma was first noted on the forehead, and this had gradually increased in size.

On physical examination the pulse was 88, respirations 20 and the blood pressure 80/60. He was poorly developed and there was marked cyanosis and clubbing. A pulsating hemangioma on the right side of the forehead was noted. The heart was not remarkable, nor were any abnormalities detected over the lung fields. Laboratory studies revealed a red blood cell count of 9.3 million, hemoglobin 25.3 gm., and a hematocrit of 79.5. An electrocardiogram revealed a right axis deviation. The femoral arterial oxygen saturation was 68 per cent. On catheterization there was no evidence of a left-to-right cardiac shunt and the right ventricular and pulmonary artery pressures were within normal limits. It was estimated that approximately 71 per cent of the right ventricular output was being shunted in some manner into the systemic circulation.

Routine chest films and roentgenoscopy were thought to reveal no significant abnormalities

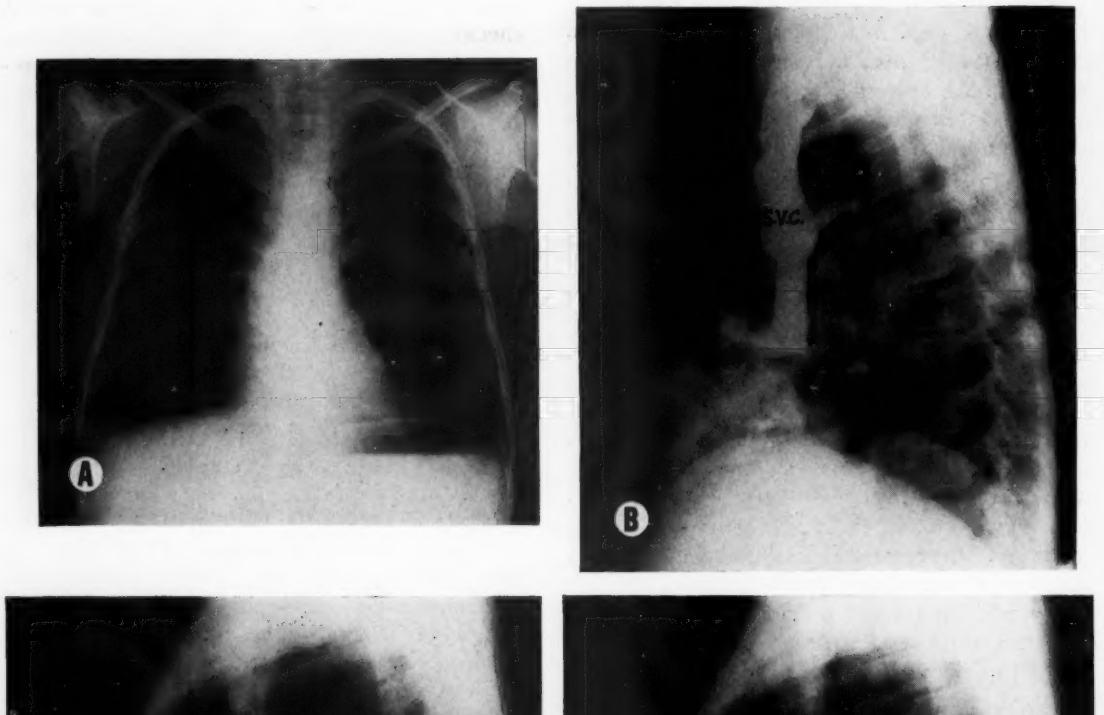

Fig. 4. Case IV. (*A*) A proved somewhat atypical arteriovenous shunt, consisting of a large vascular channel connecting the right pulmonary artery with the left atrium. Routine chest films and roentgenoscopy were thought to be normal, although in retrospect an oval prominence can be identified in the mediastinum adjacent to the right hilus.

(*B*) A lateral angiocardiogram taken at the end of one second, with the opaque medium outlining the superior vena cava (*s.v.c*) and starting to enter the right cardiac chambers.

(*C*) At the end of three seconds the right ventricular cavity and the pulmonary artery (*P.A.*) are well opacified, as is the arteriovenous shunt. There is beginning opacification of the left atrium (*L.A.*), the peripheral portions of the lung fields being abnormally avascular.

(*D*) At the end of four seconds the left-sided cardiac chambers and the aorta are well visualized. The rapid appearance of the opaque medium in the systemic circulation and the avascularity of the lung fields indicate that there is a marked shunt through the anomaly.

within the heart, great vessels or lung fields (Fig. 4*A*). It was felt clinically that the patient had an undetermined form of congenital cyanotic heart disease, and angiocardiography was requested. This examination revealed the correct nature of the process. The cardiac chambers on the right side of the heart and the main trunk of the pulmonary artery were of normal size and appearance. At the end of approximately three seconds, in both the anteroposterior and lateral projections, a peculiar, rounded opacification was noted somewhat posterior to, but in close proximity to, the right hilus. This measured approximately 1.5 cm. in diameter, and was best visualized in the lateral projection (Fig. 4*C*). Almost at the same time that the rounded area opacified, the opaque medium began to enter the left atrium, and in four seconds a normal left side of the heart and aorta were outlined (Fig. 4*D*). The peripheral portions of the lung fields at no time contained any large amount of the opaque material. The roentgenologic impression was that there was a vascular anomaly close to the right hilus permitting an arteriovenous shunt. Because of the rapid appearance of the opaque medium in the left side of the heart and the lack of opacification of the lung fields, it was felt that the amount of shunt must be quite large.

On July 14, 1948, Dr. Alfred Blalock performed a right thoracotomy. On palpating the posterior portion of the right hilus a soft pulsating mass was detected. The right pulmonary artery was exposed, and in the course of this a large thin-walled vessel was encountered arising from the posterior part of the right pulmonary artery and extending posteriorly. Further dissection revealed that it was continuous with the pulsating mass noted previously, and its distal portion seemed to enter the left atrium. This anomalous vessel was ligated and divided, whereupon the patient's color improved markedly, and it was decided that no further surgery was indicated. The postoperative course was smooth. At the time of discharge two weeks following surgery, his color appeared excellent.

One year postoperatively a check revealed that the patient was asymptomatic, and he denied exercise restriction. He had gained 16 pounds. Mild cyanosis was thought to be present, but this was questioned when an arterial oxygen saturation of 93 per cent was obtained. Blood studies revealed a red blood cell count of 4.6 million, hemoglobin of 13 gm., and a hematocrit of 39. Two and three year postoperative checks were obtained, and the patient continued to lead a normal life. Cyanosis and clubbing were no longer present, and hematologic studies were within normal limits.

Comment. This case presents several atypical features. While it was felt in retrospect that the lesion could be identified on the routine chest films, its presence was not detected prior to angiocardiography. The anatomy of the lesion was somewhat unusual in that there appeared to be an almost continuous channel between the right pulmonary artery and the left atrium, and a typical arteriovenous aneurysm was not detected. It is conceivable that this represented some other type of malformation, but from a practical standpoint its effect was exactly similar to that occurring in pulmonary arteriovenous aneurysms with large arteriovenous shunts.

Case v. F. P. (J.H.H. 423269), a white male, aged twenty-four, was admitted for the first time on May 27, 1947, for study and consideration of surgery for multiple pulmonary arteriovenous aneurysms. His family and past history were noncontributory. The present illness dated back to childhood, when cyanosis and exertional dyspnea were noted at the age of ten years. He was seen at the Graduate Hospital in Philadelphia in 1941 and again in 1944. Positive findings at that institution included cyanosis, clubbing and a systolic murmur heard over the left chest anteriorly. A small angioma was noted behind the right ear. Laboratory studies revealed considerable polycythemia, and films showed rounded densities in both lung fields. Laminagrams are said to have clearly revealed the vascular nature of the lesions. Because of the multiplicity of the lesions and because the patient was essentially asymptomatic, surgery was not recommended. In 1946 Makler and Zion[56] reported this case as an example of multiple pulmonary hemangiomata. The patient remained well until early in 1947, when he had some transitory attacks of numbness involving the left side of the face and left upper extremity, as well as episodes of syncope occurring with change in position. Because of this, the patient was referred for consideration of surgery.

On physical examination the pulse was 80, respirations 15 and the blood pressure 100/80. He was well developed and well nourished. There was moderate cyanosis and clubbing, and scattered cutaneous angiomas were noted. The heart and lungs were clear save for a to-and-fro bruit at the right base posteriorly which was maximal in systole and accentuated by deep inspiration. Laboratory studies revealed a red blood cell count of 6.9 million, hemoglobin 22.3 gm., and a hematocrit of 67. Femoral arterial oxygen saturation was 86 per cent. On catheterization the right ventricular and pulmonary artery pressures were within normal limits, and it was estimated that 42 per cent of the right ventricular output was being shunted through

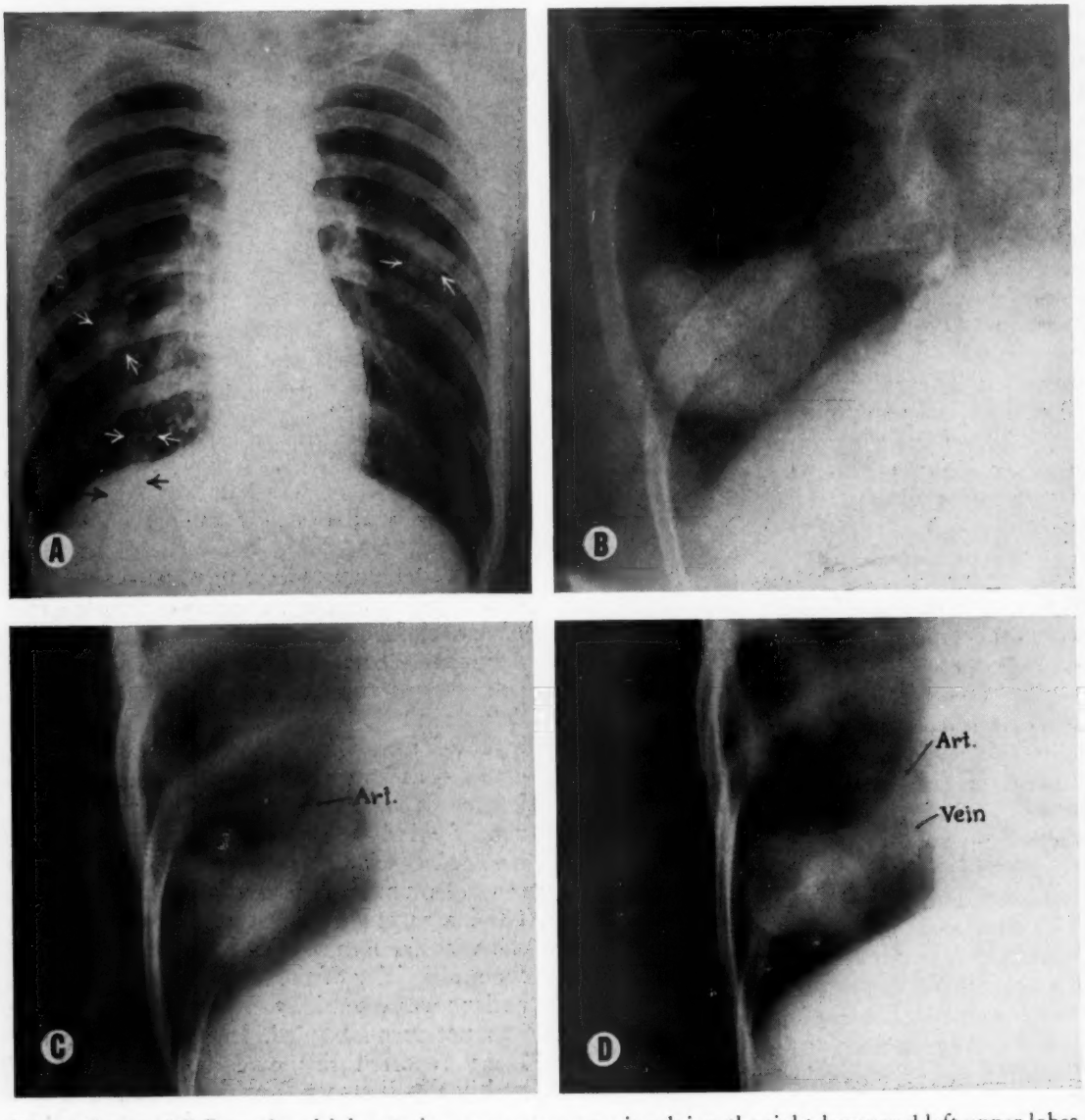

Fig. 5. Case v. (*A*) Proved multiple arteriovenous aneurysms involving the right lower and left upper lobes. A routine chest film reveals the rounded parencymal densities within both lung fields. One of the lesions is obscured behind the dome of the right diaphragm, but prominent vessels can be identified leading down to it.

(*B*) A close-up oblique view showing the rounded density at the right base posteriorly.

(*C*) and (*D*). Laminagrams at slightly different levels reveal the lobulated nature of the arteriovenous aneurysm at the right base and demonstrate clearly the prominent afferent and efferent vascular channels supplying the lesion.

the aneurysms. Routine chest films revealed two rounded densities in the right lower lobe and a third lesion in the left upper lobe anteriorly (Fig. 5*A* and *B*). Laminagrams revealed the lobulated nature of the lesions and demonstrated the enlarged arterial and venous trunks supplying them (Fig. 5*C* and *D*).

It was decided that surgery was advisable, and on July 23, 1947, Dr. Alfred Blalock performed a right thoracotomy. Two arteriovenous aneurysms were found in the lower lobe, and a lobectomy was performed. No lesions were found in the upper lobe, but one tiny angioma was detected subpleurally in the middle lobe. This was isolated in a suture ligature and was not removed. A careful study of the resected lower lobe, including injection of the vascular tree, revealed not only the two rather large arteriovenous aneurysms but also multiple smaller lesions which it was felt were too small to be detected clinically. His postoperative course was complicated by a massive atelectasis of the remaining portion of the right lung. This responded to bronchoscopy, and his course thereafter was reasonably satisfactory. At the time of his discharge two weeks postoperatively, there was no change in the degree of cyanosis, and the arterial oxygen saturation was only 83 per cent.

He returned one year later for consideration of further surgery. He had been relatively asymptomatic, but cyanosis and clubbing had persisted. On examination a continuous murmur could be heard over the left chest anteriorly. Laboratory studies revealed a red blood cell count of 5.6 million and a hemoglobin of 17.5 gm., representing some decrease in the degree of polycythemia, but the femoral arterial oxygen saturation was only 85 per cent. On roentgenography the arteriovenous aneurysm in the left upper lobe had not changed significantly, and no new lesions were detected. On July 26, 1948, Dr. Blalock performed a left thoracotomy. A large arteriovenous aneurysm was found in the lower third of the left upper lobe, and a segmental resection of the upper lobe was undertaken. No other aneurysms were detected in the remainder of the upper or lower lobes. His postoperative course was reasonably smooth, and he was discharged on the fifteenth postoperative day.

Letters from the patient and his physician approximately three and one-half years following his second operation stated that there was no cyanosis or clubbing, or symptoms of any sort. The red blood cell count was 4.5 million, the hemoglobin 16 gm.

Comment. This is an example of multiple symptomatic pulmonary arteriovenous aneurysms with involvement of both lungs. An apparent clinical cure followed a two-stage surgical removal of the lesions. At the time of the first operation additional small angiomas were found, these not having been detected clinically before that time, and there was no dramatic improvement in the clinical or laboratory findings between the two operations.

Case vi. M. F., a white female, aged forty, was admitted to the Sinai Hospital of Baltimore, Maryland, on January 16, 1950, for evaluation of her cardiovascular status. The family history revealed that the patient's father had frequent epistaxes and was anemic, and there was a familial history of "clubbed thumb." The patient had always been rather "sickly," and cyanosis had been first noted at the age of sixteen years. At twenty-two years of age she had an episode characterized by dyspnea, orthopnea, ankle edema and swelling of the abdomen. She was told she had heart trouble, and was put to bed for three weeks

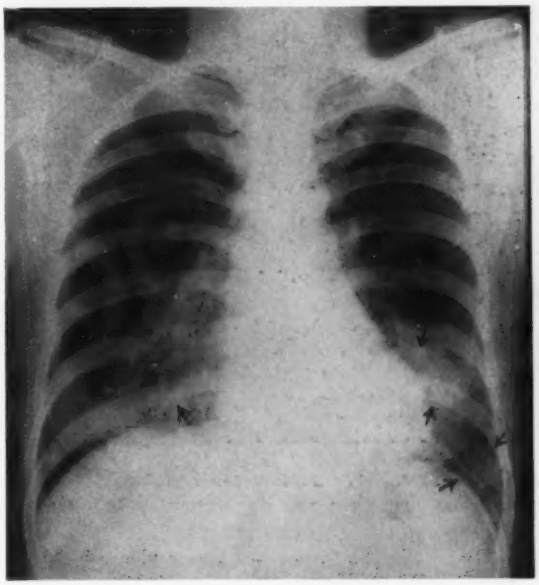

Fig. 6. Case vi. Proved multiple arteriovenous aneurysms involving both lower and right middle lobes. The rounded nature of the lesions are well shown, but the vascular channels supplying the aneurysms are not particularly prominent.

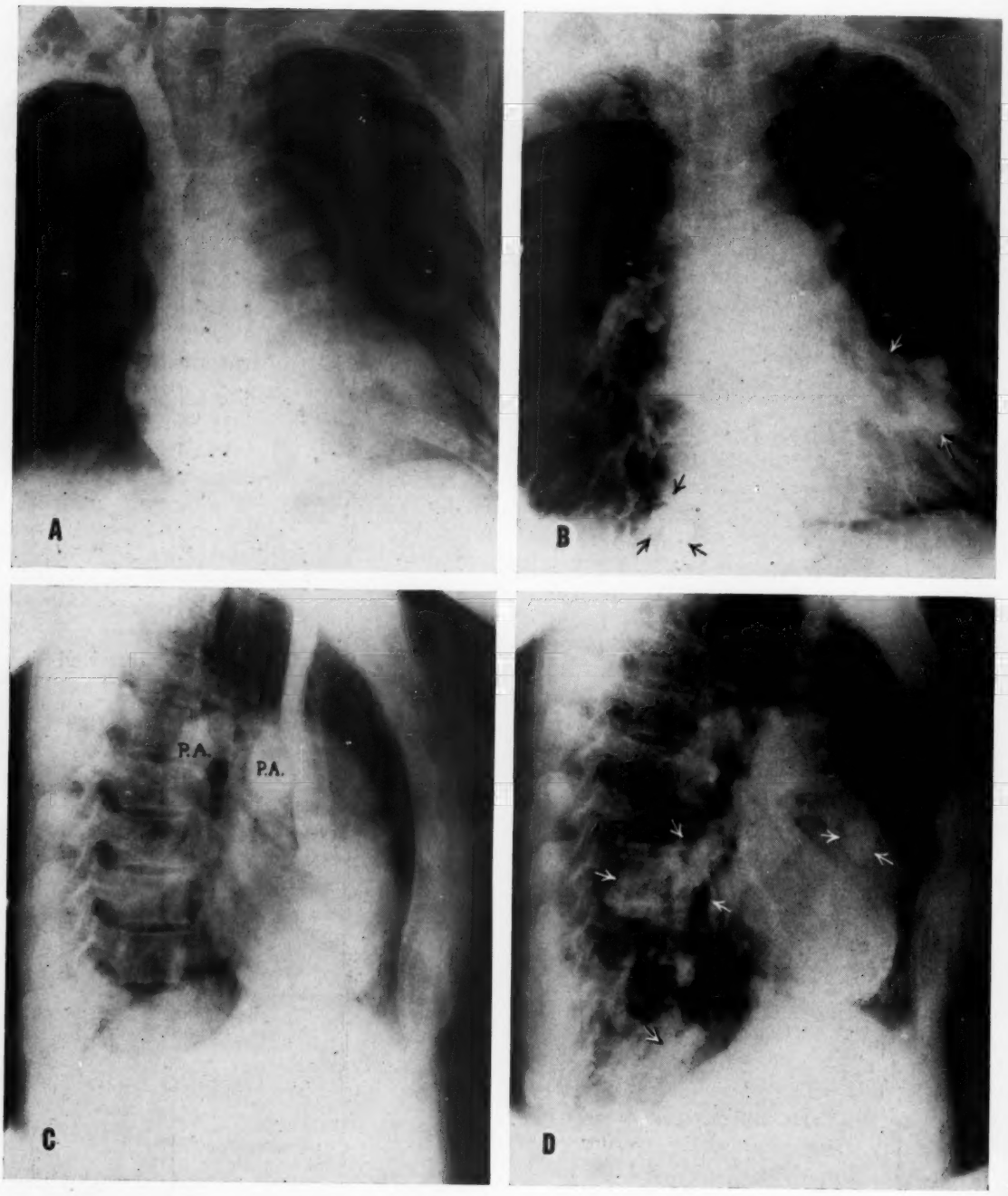

Fig. 7. Case VI. (*A*) An anteroposterior angiocardiogram taken at the end of one second, showing an opacified superior vena cava and right atrium. Note the soft tissue density of one of the arteriovenous aneurysms adjacent to the left heart border.

(*B*) A film at the end of three seconds, showing the definite opacification of the aneurysm adjacent to the left heart border. The smaller arteriovenous aneurysm in the right cardiophrenic angle is partially obscured.

(C) A lateral film at the end of one second, demonstrating filling of the superior vena cava, right side of the heart, pulmonary artery and its branches with the opaque medium.

(*D*) A film at the end of three seconds demonstrating the opacification of the vascular channels within and adjacent to the arteriovenous aneurysms.

with apparent recovery. Intermittently during the next eighteen years there were episodes of heart failure, usually responding to bed rest and digitalization. Shortly before admission, however, she had a severe episode of orthopnea and exertional dyspnea, along with ankle edema and swelling of the abdomen. This did not respond to the usual therapy, and she was admitted for further study.

Physical examination on admission revealed a pulse of 97, respirations 24, and blood pressure 140/70. There was moderate respiratory distress. A noticeable pallor was present, as well as definite cyanosis and clubbing. Telangiectasis were noted on the tongue. The heart was enlarged, with systolic and diastolic precordial murmurs. Moist rales were present at the lung bases, and minimal presacral edema was noted. Initial laboratory studies revealed a rather marked anemia, with a red blood cell count of 3.3 million, hemoglobin 5.5 gm., and a hematocrit of 30. An electrocardiogram revealed right heart strain. Chest films revealed some cardiac enlargement, with mottled densities at both lung bases (Fig. 6). The largest of these was situated in the left lower lobe, and measured roughly 3 cm. in diameter.

The admission impression was probable rheumatic heart disease with decomposition, but on further evaluation the possibility of pulmonary arteriovenous aneurysms was considered, and the patient was referred to The Johns Hopkins Hospital for catheterization and angiocardiography. The former revealed right ventricular and pulmonary artery pressures barely above the upper limits of normal. The brachial arterial oxygen saturation was 68.8 per cent, and it was estimated that 62 per cent of the right ventricular output was being shunted through the hemangiomas. The angiocardiographic study revealed typical arteriovenous aneurysms in both lower lobes, and in the middle lobe as well (Fig. 7*A*, *B*, *C* and *D*). The main trunk of the pulmonary artery and its hilar divisions were not dilated. The tortuous channels within the aneurysms began to fill promptly, and at the end of three to four seconds there was initial opacification of the left side of the heart and aorta.

During the first part of her hospital stay, she was treated for congestive failure, and received several transfusions. Two months following admission, her hematological status had changed considerably, the red blood cell count being 6.6 million, the hemoglobin 15.4 gm., and the hematocrit 59. She ran a spiking fever, the cause of which was not determined.

On March 24, 1950, a left thoracotomy was performed at Sinai Hospital by Dr. W. Rienhoff. Several arteriovenous aneurysms were found in the lower lobe, and a left lower lobectomy was undertaken without difficulty. Study of the resected lobe confirmed the operative impression, and in one of the arteriovenous aneurysms there was a projecting but adherent thrombotic mass measuring 2.5 by 1.2 by 1.5 cm. Her postoperative course was uneventful.

Two weeks postoperatively the cyanosis was decreased but still present, and blood studies revealed moderate polycythemia. She was recatheterized, and on this occasion the right ventricular and pulmonary artery pressures were within normal limits. The peripheral arterial oxygen saturation had risen to 77.6 per cent, and it was estimated that now only 51 per cent of the right ventricular output was returning unsaturated to the left side of the heart.

Three weeks following the first operation, Dr. Rienhoff performed a right thoracotomy. The right lower lobe was removed with difficulty, there being considerable hemorrhage during the operation. The removed specimen revealed several typical arteriovenous aneurysms. The patient did not do well postoperatively, and death occurred approximately twenty hours later. At autopsy there was dilation and hypertrophy of the right atrium and ventricle. The right upper lobe was atelectatic, the middle infarcted. It was thought that death was secondary to pulmonary insufficiency, since the left upper lobe was the only functioning segment of pulmonary tissue following the second operation.

Comment. This is another example of multiple symptomatic arteriovenous aneurysms with involvement of both lungs. Initially a profound anemia masked the expected polycythemic response. The cause of this anemia and its relationship to the episodes of congestive failure are not clear. A two-stage surgical removal of the lesions was undertaken, with death occurring in the immediate postoperative period following the second thoracotomy.

Case vii. C. S. (J.H.H. A73497), a white female, aged eight, was admitted for the first

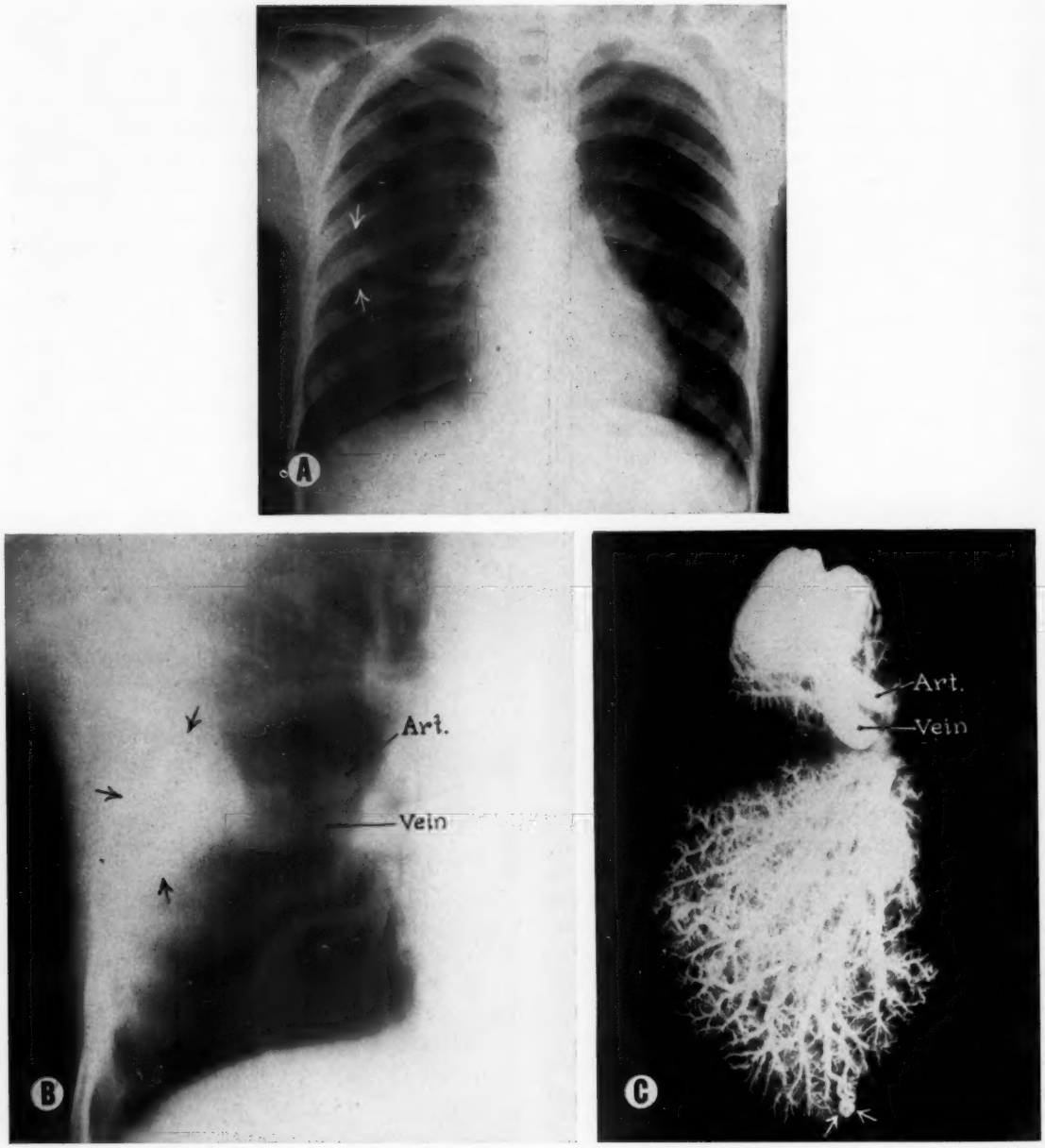

Fig. 8. Case vii. (*A*) A proved arteriovenous aneurysm involving the right middle lobe, with additional multiple small angiomas not suspected prior to surgery. A routine film reveals the rounded density of the largest aneurysm in the lateral aspect of the right mid-lung field.

(*B*) A laminagram clearly reveals the rounded arteriovenous aneurysm and two large vessels extending into it from the hilus.

(*C*) The arterial tree of the resected right middle lobe was injected with a barium suspension. The specimen film reveals the large arteriovenous aneurysm, as well as the enlarged afferent and efferent vascular trunks supplying it. One of the smaller arteriovenous shunts is demonstrated at the base. It is questionable whether lesions of this small size can be demonstrated by roentgenographic means. The triangular area in which the vascular tree is not filled represents an injection artefact.

time in October, 1949, for evaluation of cyanosis and polycythemia. The family history was of interest in that the father has an unproved but probable pulmonary arteriovenous aneurysm (see Case VIII). The present illness was of at least a year's duration, the parents having noticed cyanosis and chronic fatigability during that time. There had been no observed dyspnea, epistaxes, hemoptyses or neurological disturbances.

On physical examination the pulse and respirations were normal, and the blood pressure was 90/60. There was definite cyanosis and clubbing. The heart and lungs were unremarkable, no pulmonary murmur being detected, nor were any telangiectases of the skin or mucous membranes noted. Laboratory studies revealed a red blood cell count of 8.4 million, hemoglobin 17.5 gm., and a hematocrit of 53.2. The systemic arterial oxygen saturation was 77 per cent. On catheterization, the right ventricular and pulmonary artery pressures were within normal limits, and it was estimated that 61 per cent of the right ventricular output was being returned unoxygenated to the left side of the heart.

Routine chest films revealed a rounded homogeneous density in the lateral aspect of the right mid-lung field, with minimal infiltration extending from the medial portion of the lesion to the right hilus (Fig. 8*A*). The lungs were thought to be otherwise clear, and the heart and great vessels were unremarkable in appearance. The initial roentgenologic impression was that the lesion represented a primary tuberculous process. The tuberculin test was negative, however, and because of this and the findings of cyanosis and polycythemia, the possibility of a pulmonary arteriovenous aneurysm was considered clinically. Laminagrams revealed the vascular nature of the process, showing a sharply defined rounded density in the lateral aspect of the right middle lobe, with at least two dilated vascular channels leading into the right hilus (Fig. 8*B*). No definite lesions were detected elsewhere in the lungs.

On October 29, 1949, Dr. M. Ravitch performed a right thoracotomy. A bluish varicose tumor with vigorous pulsations was found in the lateral aspect of the middle lobe. In addition, all over the upper and lower lobes there were innumerable tiny angiomas varying from 2 or 3 mm. to 1 or 1.5 cm. in diameter. Most of these seemed to be no more than telangiectases, but at least one on the upper lobe was slightly raised and could be palpated with the finger as being firmer than the surrounding lung. Because it was suspected that there might be similar multiple small lesions in the left lung, it was elected to perform a right middle lobectomy. Roentgenograms of the specimen following injection of the vascular tree with opaque medium revealed the larger lesion detected clinically, as well as several smaller arteriovenous aneurysms (Fig. 8*C*).

The patient's postoperative course was uneventful. She was still slightly cyanotic, and study revealed that the shunt through the remaining lesions was in the neighborhood of 45 per cent. On examination two years after surgery, there was mild but definite cyanosis with minimal clubbing, and the red blood cell count was 5.3 million.

Comment. This is a proved case of pulmonary arteriovenous aneurysms in a patient whose father apparently has the same anomaly. Of chief interest was the finding at surgery of numerous small angiomas whose presence had not been detected clinically prior to that time. In retrospect, the vascularity of the lung fields may be somewhat increased on the routine chest films and laminagrams, but certainly no discrete lesions can be identified except for the obvious one in the right middle lobe. It is problematical whether angiocardiography would have revealed their presence. Also of interest is the fact that anoxemia persisted following the surgical removal of the single gross arteriovenous aneurysm. This would tend to support the concept that the summation of the minute arteriovenous shunts through many small angiomas may be enough to produce anoxemia with resultant cyanosis and polycythemic response.

Case VIII. T. S. (J.H.H. 519295), a white male, aged forty, was the father of the patient in Case VII. Because of his daughter's history, he was asked to come in for a routine examination. Past history was unremarkable save for episodes of epistaxes dating back many years, and he stated that his father had a similar history. There was no present illness, the patient having no symptoms suggesting the presence of a pulmonary arteriovenous aneurysm. He had always led a normal, active life.

On physical examination the pulse was 72 and respirations were 14. There was no obvious cyanosis, and the mucous membranes were of good color. There was suggestive clubbing. Numerous dilated arterioles were noted over the nose and eyelids. The heart and lungs were unremarkable, no pulmonary murmurs being detected. Laboratory studies revealed a red blood cell count of 5.1 million, hemoglobin 13 gm., hematocrit of 42, and a white blood cell count of 10,200, with a normal differential count. A systemic arterial oxygen saturation was reported as 98 per cent.

On routine chest films the heart and great

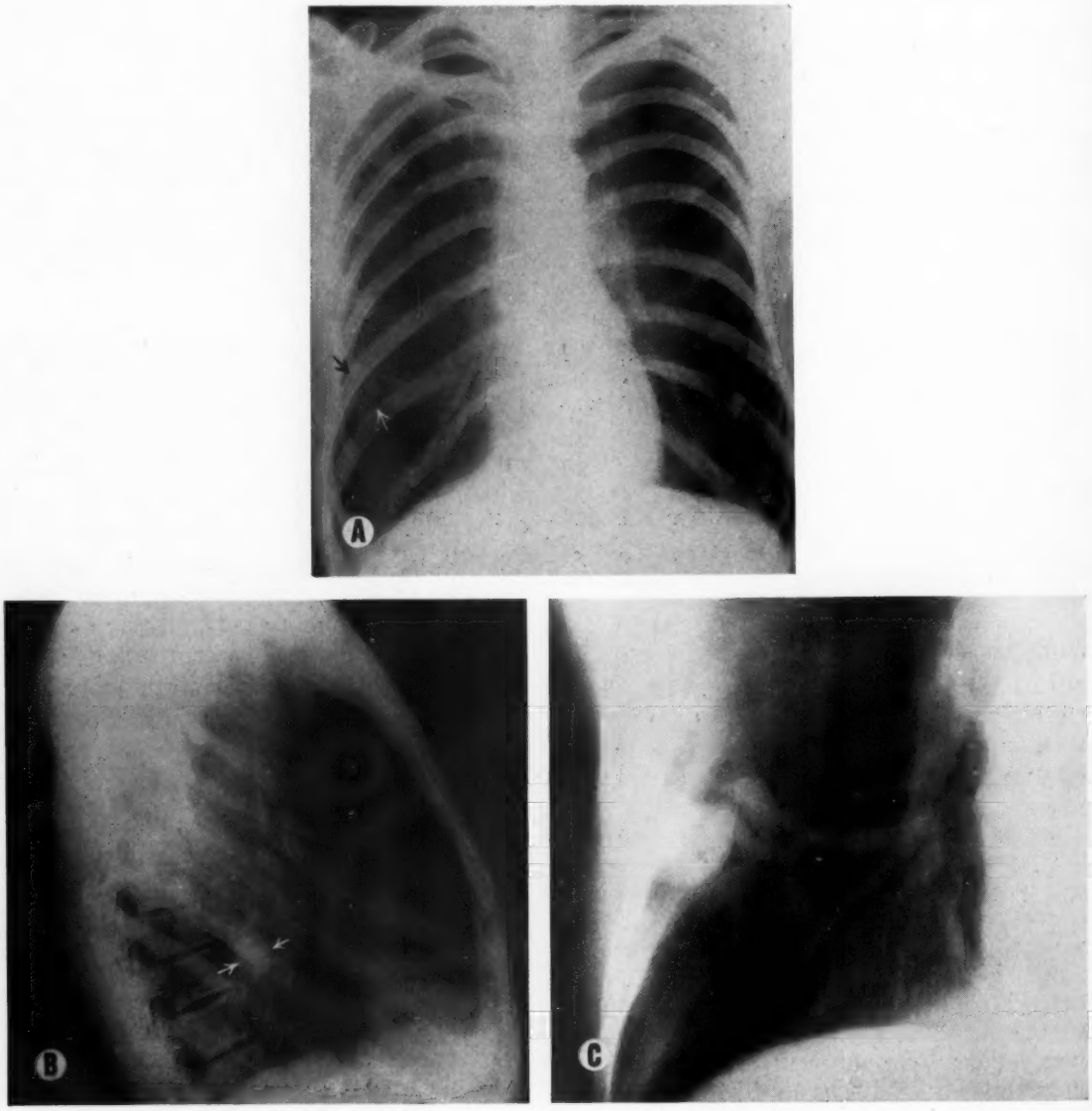

Fig. 9. Case VIII. (*A*) An unproved but probable arteriovenous aneurysm, discovered in the father of the patient in Case VII. The routine posteroanterior projection reveals a rather indistinct density in the lateral aspect of the right midlung field.

(*B*) A lateral projection reveals the lesion more clearly, and demonstrates that it is in the right lower lobe.

(*C*) A laminagram reveals the rounded nature of the aneurysm, and demonstrates only one prominent vascular channel connecting it to the hilus. This is of interest in view of the fact that there was no clinical or laboratory evidence of a significant arteriovenous shunt in this patient.

vessels were within normal limits. On the posteroanterior projection a rather poorly defined area of increased density was noted in the lateral aspect of the right mid-lung field (Fig. 9*A*). In the lateral projection it was more distinct, and was seen to be within the lower lobe (Fig. 9*B*). Another smaller rounded density was noted in the left costophrenic angle. Laminagrams of the lesion in the right lower lobe revealed a somewhat lobulated area of fairly homogeneous density in maximal focus at 9 cm. from the posterior chest wall. Only one abnormally large vascular channel, which in its peripheral portion divided into several branches, was noted entering the area of increased density (Fig. 9*C*). Laminagrams of the left costophrenic angle were not satisfactory. It was the roentgenologic impression that the lesion in the right lower lobe represented a pulmonary arteriovenous aneurysm.

Because the patient was totally asymptomatic and since there was no evidence of a shunt, it was felt that surgical intervention was not justified.

Comment. This case is presented as an unproved but probable arteriovenous aneurysm in an individual whose daughter had proved pulmonary arteriovenous aneurysms. The patient was totally asymptomatic, without evidence of an arteriovenous shunt through the lesion. The correct nature of the process would not have been suspected from routine films in the absence of the positive family history. It is of interest that the laminagrams revealed only one enlarged vascular channel supplying the lesion, since in the usual case both afferent and efferent trunks can be identified.

Case IX. A. G. (J.H.H. 558783), a white male, aged thirty-eight, was admitted to the urological service on February 9, 1952, for the removal of penile warts. The family and past histories were noncontributory. During the routine examinations cyanosis and polycythemia were detected. Careful questioning revealed that the patient's lips had been bluer than normal since childhood, and that he had been rejected by selective service in 1945 because of a "spot on the lung." He had always been active, without exertional dyspnea, hemoptysis or other symptoms.

On physical examination the pulse was 100, respirations were 14, and the blood pressure was 105/65. There was mild cyanosis, but the presence or absence of clubbing is not specifically mentioned in the chart. No cutaneous or mucosal telangiectases were observed. The heart and lungs were unremarkable, no pulmonary murmurs being detected. The spleen was not enlarged. There were several wart-like growths on the penis. Laboratory studies revealed a red blood cell count of 5.9 million, hemoglobin 20 gm., and a hematocrit of 61.5. The femoral arterial oxygen saturation was found to be 82 per cent, and with the dye dilution test it was estimated that there was a right-to-left shunt of approximately 15 per cent of the cardiac output. An electrocardiogram was normal.

Routine films revealed a normal appearing heart. The right hilar region was somewhat prominent, and there was an oval density measuring roughly 1.5 by 4 cm. immediately adjacent to the upper border of the right hilus (Fig. 10*A*). It was possible to obtain the chest film taken seven years previously during the selective service examination, and roentgenologically the lesion had not changed during that period. At roentgenoscopy there were moderate pulsations in the right hilar area and adjacent density. Laminagrams of this region revealed the soft tissue mass to be composed chiefly of a single somewhat coiled tube-like structure (Fig. 10*B*). Angiocardiography was performed. The right atrial and ventricular chambers were normal. The main trunk of the pulmonary artery was not ideally visualized, but the right main pulmonary artery was well demonstrated and was dilated. The density above the right hilus clearly opacified, and its appearance suggested a single large coiled vessel. There were, in addition, some abnormal vascular contours within the right hilus itself which were felt to be part of the malformation (Fig. 10*C* and *D*). A moderate amount of opaque medium passed out into the peripheral vessels of both lungs, and no other vascular abnormalities were detected. The contrast medium did not appear in the left side of the heart with undue rapidity.

During the course of these studies, the penile warts were surgically removed. There was considerable discussion concerning the advisability of exploratory thoracotomy for the obvious pulmonary arteriovenous aneurysm. It was generally felt that surgery was indicated, but because of the asymptomatic nature of the lesion and the patient's reluctance to undergo surgery, it was decided that such a course could

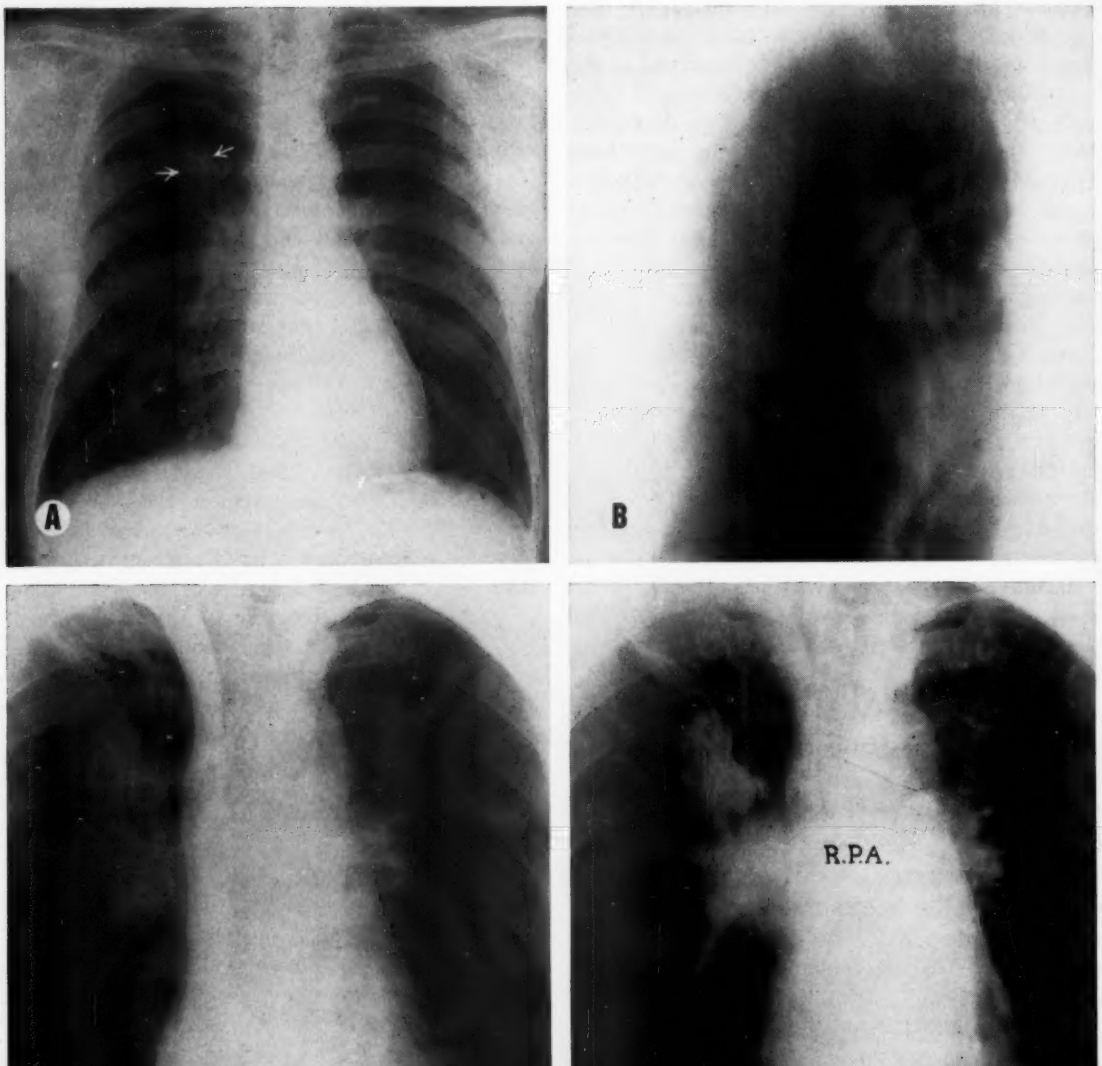

Fig. 10. Case IX. (*A*) An unproved but probable arteriovenous aneurysm adjacent to the upper border of the right hilus. A routine film reveals the oval soft tissue density, as well as a somewhat prominent right hilus.

(*B*) A laminagram reveals the oval soft tissue density to be composed of a single coiled tube-like structure.

(*C*) An anteroposterior angiocardiogram at the end of one second reveals an opacified superior vena cava and right atrium. The contrast medium has not reached the pulmonary artery.

(*D*) A film at the end of three seconds reveals the opacification of the soft tissue density adjacent to the right hilus, and suggests that it is a single large, coiled vessel. Of interest is the fact that the right pulmonary artery (*R.P.A.*) is moderately dilated.

not be strongly urged. It was eventually decided to observe the patient for a period of time, and he was discharged from the hospital.

Comment. The presence of an unproved but probable pulmonary arteriovenous aneurysm was detected accidentally during a hospital admission for other reasons. While there was clinical evidence of an arteriovenous shunt, the patient was totally asymptomatic and surgery was not strongly recommended.

VI. SUMMARY

Congenital arteriovenous aneurysms of the lung are relatively uncommon, but have been reported with increasing frequency during the past decade, presumably because of an increased interest in and clinical awareness of the anomaly. The pathological anatomy and disturbed physiology associated with pulmonary arteriovenous aneurysms are discussed. Fundamentally it is felt that these lesions are hemangiomatous in nature, the majority being cavernous-type hemangiomas with large vascular channels permitting varying degrees of arteriovenous shunt. Physiological disturbances, when present, are secondary to the shunting of unoxygenated blood back into the left side of the heart with resultant anoxemia and a polycythemic response of varying intensity.

The clinical aspects of the anomaly are presented, based upon a review of 85 cases reported in the literature, and include analyses of symptomatology, physical findings, laboratory data, roentgenologic findings, treatment and outcome. Symptoms, when present, are usually due to the anoxemia and polycythemia and less commonly to the arteriovenous aneurysm itself. Many of the abnormal physical and laboratory findings are also secondary to the anoxemia, the only positive physical finding in conjunction with the aneurysm itself being the presence of a vascular murmur heard through the overlying lung and chest wall. Large lesions are easily demonstrable on routine roentgenography, but small arteriovenous aneurysms and telangiectases can probably not be demonstrated by roentgenologic means. Laminagraphy and angiocardiography are the most definitive roentgenologic studies available for accurate identification of pulmonary arteriovenous aneurysms. Treatment, when indicated, consists of surgical removal of the aneurysm.

Nine cases of pulmonary arteriovenous aneurysms which have been seen clinically at The Johns Hopkins Hospital are presented. Seven of these have been proved, and in the remaining two cases the diagnosis seems reasonably certain. Two patients were members of the same family.

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THE ROENTGENOGRAPHIC ASPECTS OF THROMBOSIS OF ANEURYSMS OF THE ANTERIOR COMMUNICATING AND ANTERIOR CEREBRAL ARTERIES*

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THE angiographic diagnosis of intracranial aneurysms is firmly established. Many communications describing their characteristic appearance have appeared in the radiologic and neurosurgical literature. Reports of closure of such aneurysms by thrombus are rare. List and Hodges[2] stated that there were instances in which the diagnosis of an intracranial aneurysm was likely from a clinical viewpoint, but angiographic verification was unobtainable because the small size of the neck of the aneurysm prevented entry of the contrast substance, or the aneurysm was filled with hematoma. No specific examples were cited by these authors. Löfstedt[3] observed that none of the 17 aneurysms seen by him recently were thrombosed, and went on to say that "on critically reading through the literature one finds very few actually proved thrombosed aneurysms." As far as I could determine there have been no reports of cases in which thrombosis of intracranial aneurysms were demonstrated roentgenologically.

The present case of a patient with an aneurysm of the left anterior communicating artery at its junction with the left anterior cerebral artery is of interest because the aneurysmal sac was plugged with thrombus which extended into and blocked the anterior cerebral artery. Complete roentgenologic data were available and necropsy confirmation was obtained. Of further interest are 2 other patients with essentially similar complaints and almost identical angiographic and pneumoencephalographic findings. Neither of the latter two were operated upon, and both improved clinically so that verification was not obtained.

REPORT OF CASES

CASE I. L. B., a man, aged thirty-eight, complained of bilateral severe headache unaccompanied by other symptoms for two months. This was followed by a sudden episode of unconsciousness following a bowel movement at which the patient said he did not strain. He was unconscious then for about nine hours. A spinal tap done by his physician at that time was reported as having shown free blood in the cerebrospinal fluid. A cerebral angiography performed at another hospital was unsatisfactory and nothing positive was reported. He returned to work but the headaches continued and were accompanied by pain in both eyes, worse on the left side. The pain appeared to be underneath the eyeball. He complained of a sensation of grittiness in the lateral canthus of the left eye, and felt that that eye was closing. The patient then noted a blind spot first in the lateral corner of the left visual field, later extending to the center of the field. He was completely blind in the left eye for one week before entering the hospital. There was no involvement of the other cranial nerves, nor were there any motor or sensory changes.

Physical examination showed both ocular fundi to be normal. The right eye accommodated to light and accommodation, while the left eye did not react directly to light but did so consensually. There was no ptosis of either eyelid, and the movements of the eyes were normal. The eye consultant reported that in his opinion there was a retrobulbar lesion somewhere between the bulb and the optic chiasm.

Plain roentgenograms of the skull showed the bony structures to be normal. No unusual calcific shadows were present within the cranial cavity.

A left cerebral angiogram in the lateral position showed no filling of the anterior cerebral artery. The point of block was sharp, and no sac-like collection of contrast medium was present at or near this point, which appeared to be

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the junction of the left anterior communicating and anterior cerebral arteries. The ophthalmic artery filled unusually well and could be followed well into the orbit. The middle cerebral group was elevated somewhat, but otherwise was not distorted. The carotid syphon was intact.

A left frontal angiogram showed no diodrast in the anterior cerebral artery, while the middle cerebral system filled well. No aneurysmal pouch was visible.

It was believed that there might be a mass lesion occluding the anterior cerebral artery. A pneumoencephalogram was made the next day. This revealed the lateral ventricles to be normal in size, contour and position. The third

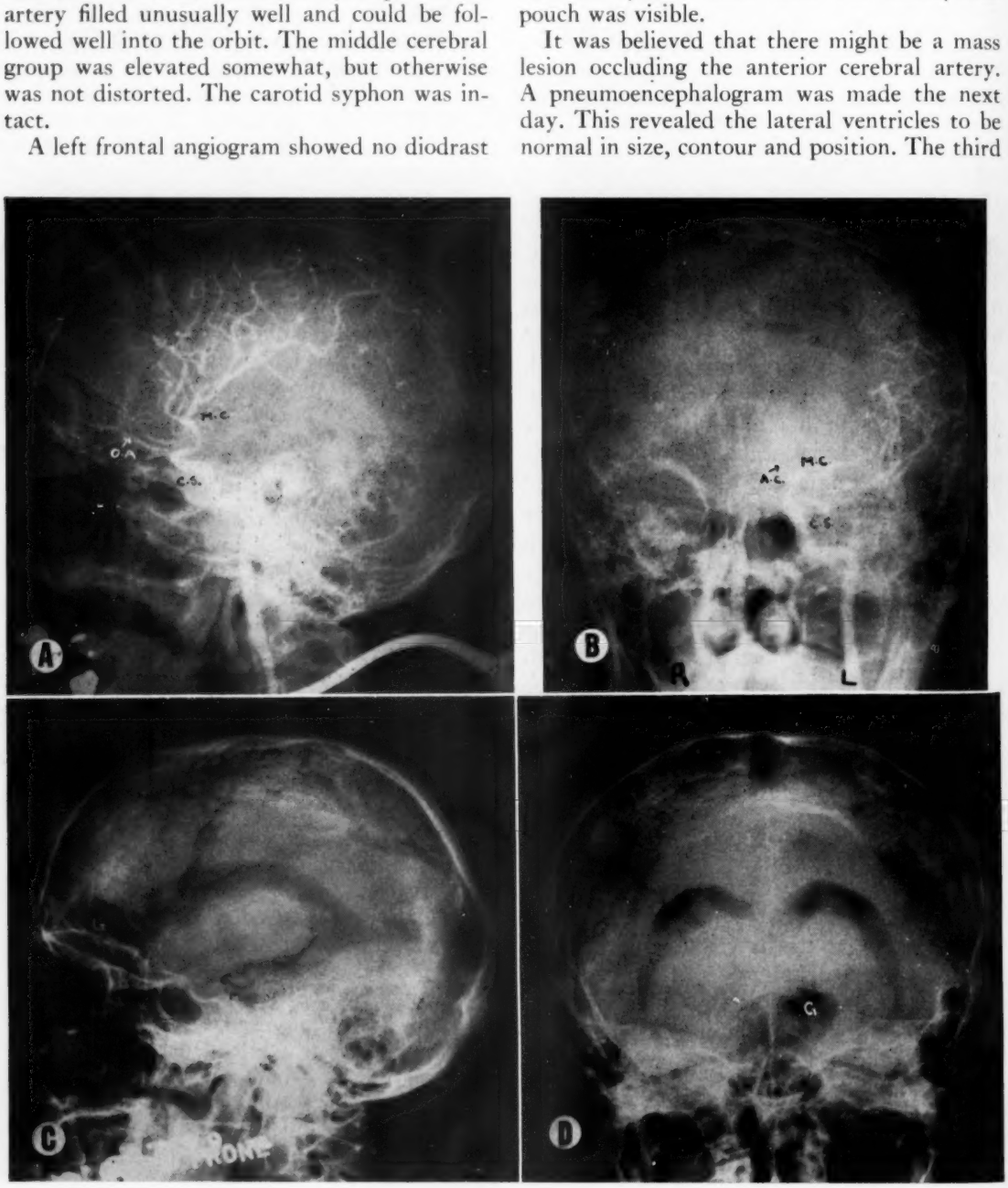

Fig. 1. Case 1. (*A*) Left lateral cerebral angiogram, arterial phase. The middle cerebral system (M.C.) is well filled and is displaced upwards to a slight degree. The ophthalmic artery (O.A.) is well filled. The carotid syphon (C.S.) is normal. The anterior cerebral artery is not visualized. (*B*) Anteroposterior angiogram, arterial phase. The carotid syphon (C.S.) and middle cerebral arteries (M.C.) are well filled and in their normal situations. The anterior communicating artery (A.C.) is present as a faint stump. The anterior cerebral artery is not filled. (*C*) Right prone lateral pneumoencephalogram. The ventricles are normal. Gas has escaped into the left subfrontal space (G). The cisterna chiasmatis is obliterated by a soft tissue mass (M), while just posterior the cisternae interpeduncularis and pontis are normal. (*D*) Anteroposterior

and fourth ventricles were not remarkable. A soft tissue shadow was present bulging into the cisterna chiasmatis with a dorsal convexity. The cisternae interpeduncularis and pontis were well filled and were normal. Of great interest was the appearance of gas in the left subfrontal region later in the course of the pneumoencephalographic examination. This gas did not appear on the first films but could be seen on the roentgenograms obtained about ten minutes after the lumbar insufflation had been completed.

These observations were interpreted as indicating a mass lesion in the vicinity of the optic chiasm together with disruption of the cortex of the inferior surface of the left frontal lobe which permitted gas to seep through the diseased or destroyed brain tissue into the subfrontal subarachnoid region.

The patient became comatose and had a

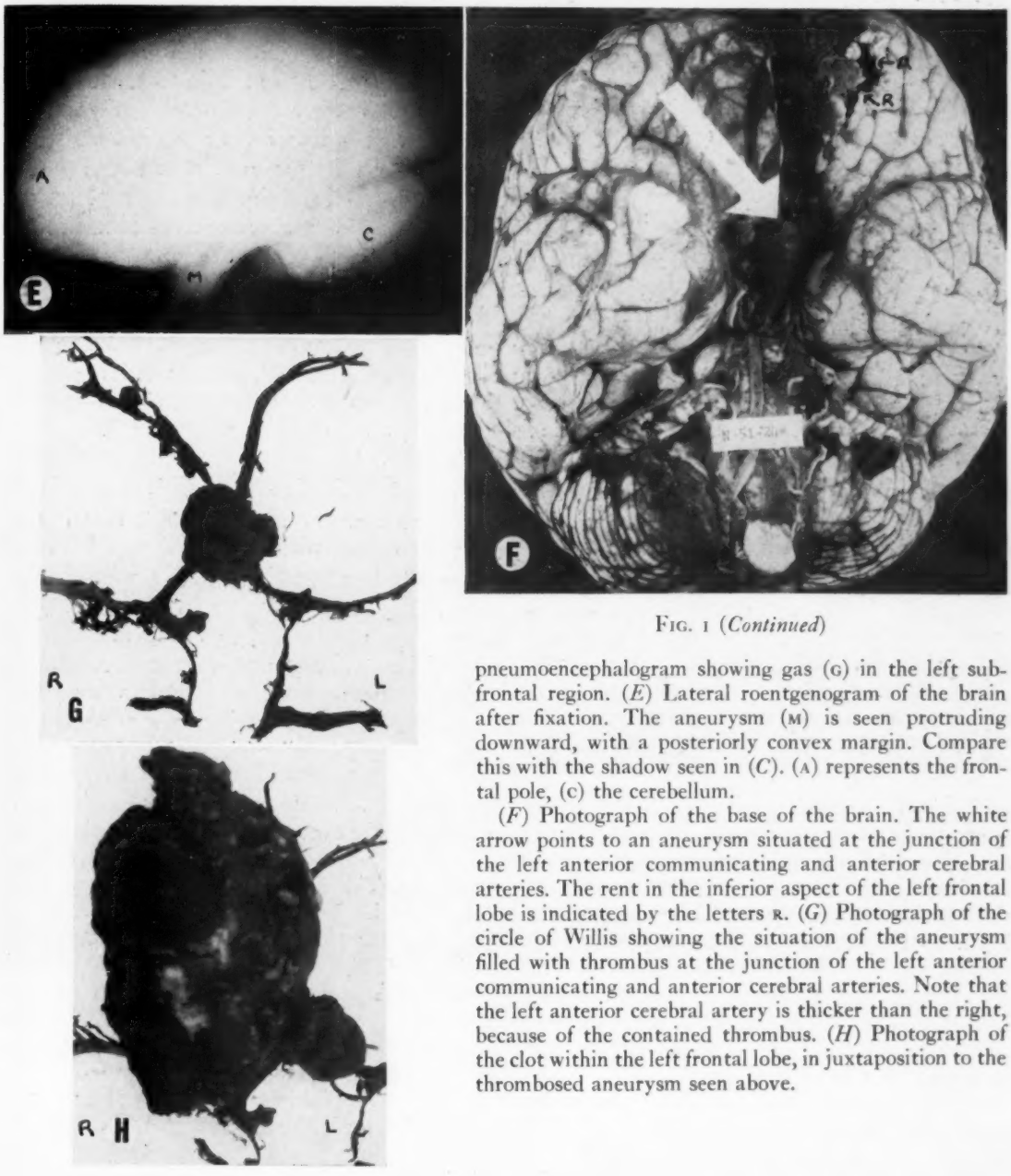

Fig. 1 (*Continued*)

pneumoencephalogram showing gas (G) in the left subfrontal region. (*E*) Lateral roentgenogram of the brain after fixation. The aneurysm (M) is seen protruding downward, with a posteriorly convex margin. Compare this with the shadow seen in (*C*). (A) represents the frontal pole, (C) the cerebellum.

(*F*) Photograph of the base of the brain. The white arrow points to an aneurysm situated at the junction of the left anterior communicating and anterior cerebral arteries. The rent in the inferior aspect of the left frontal lobe is indicated by the letters R. (*G*) Photograph of the circle of Willis showing the situation of the aneurysm filled with thrombus at the junction of the left anterior communicating and anterior cerebral arteries. Note that the left anterior cerebral artery is thicker than the right, because of the contained thrombus. (*H*) Photograph of the clot within the left frontal lobe, in juxtaposition to the thrombosed aneurysm seen above.

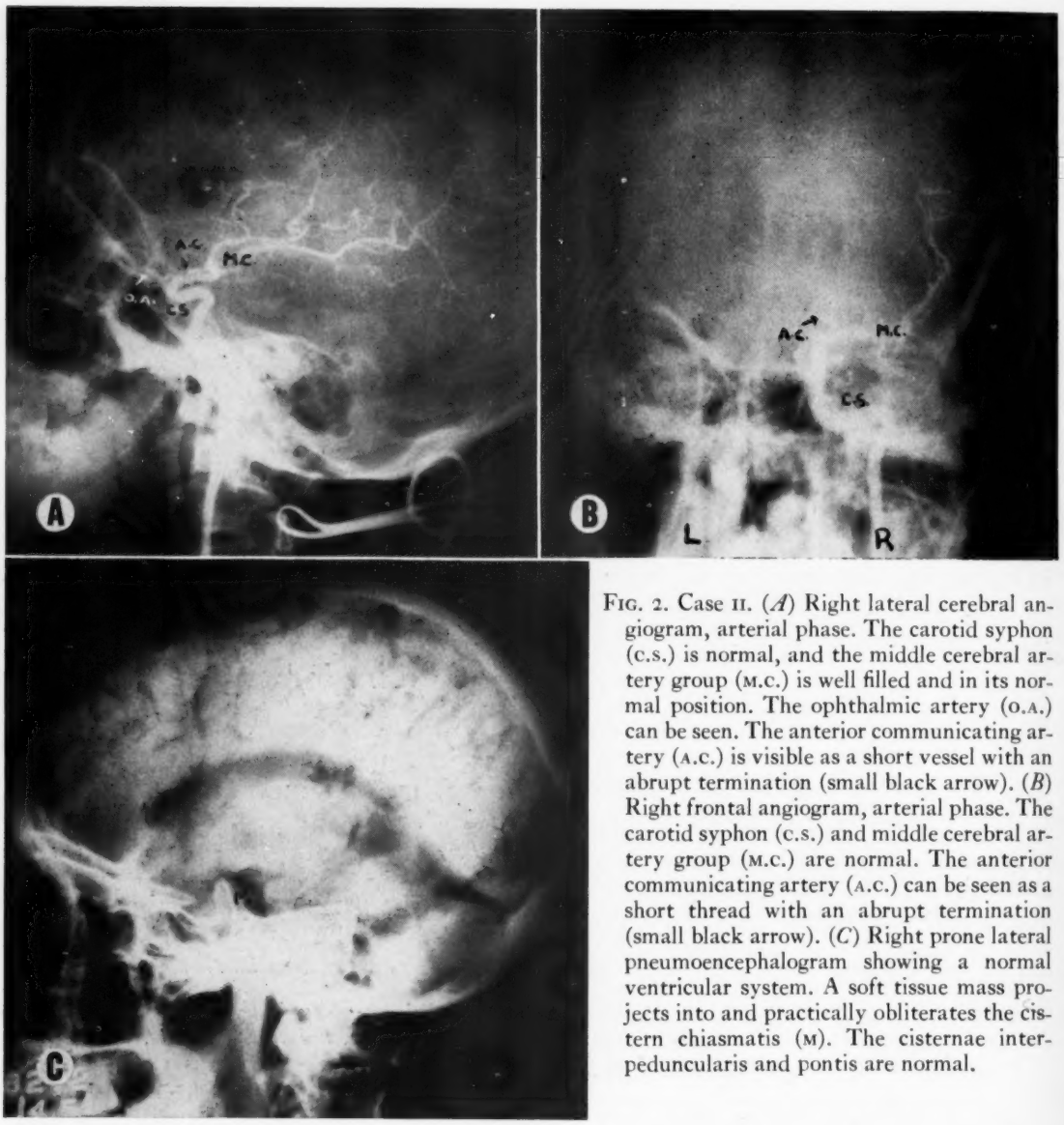

Fig. 2. Case II. (*A*) Right lateral cerebral angiogram, arterial phase. The carotid syphon (C.S.) is normal, and the middle cerebral artery group (M.C.) is well filled and in its normal position. The ophthalmic artery (O.A.) can be seen. The anterior communicating artery (A.C.) is visible as a short vessel with an abrupt termination (small black arrow). (*B*) Right frontal angiogram, arterial phase. The carotid syphon (C.S.) and middle cerebral artery group (M.C.) are normal. The anterior communicating artery (A.C.) can be seen as a short thread with an abrupt termination (small black arrow). (*C*) Right prone lateral pneumoencephalogram showing a normal ventricular system. A soft tissue mass projects into and practically obliterates the cistern chiasmatis (M). The cisternae interpeduncularis and pontis are normal.

generalized convulsion, and died about ten hours later. Necropsy revealed a thrombosed aneurysm of the junction of the anterior communicating and anterior cerebral arteries on the left side, containing a rubbery clot measuring 2 by 1 by 0.8 cm. which completely obliterated the sac. The thrombus extended into the anterior cerebral artery completely blocking its lumen. The frontal lobe of the brain was soft and infarcted, and its inferior surface was lacerated by a large firm clot which measured 6 by 3 by 3 cm. Free blood filled the entire ventricular system.

Case II. L. L., a woman, aged thirty-six, complained of double vision accompanied by a dull headache over the right orbit for about ten days. She had had occasional nausea but no vomiting. The pain was intermittent at first, but later became constant and radiated inferiorly over the right preauricular area and right cheek and sometimes to the supraorbital area. She also noted facial paresthesia on the right side.

Physical examination showed both pupils to react normally to light and accommodation. There was no diplopia when gazing at a single object like a pin, but the patient said she saw the examiner double. There was no gross diminution of visual acuity. The right eye was

unable to rotate laterally beyond the midline.

Plain roentgenograms of the skull were normal. A pneumoencephalogram showed the ventricular system to be normal in size, contour and position. The cisterna chiasmatis was obliterated by a soft tissue mass with a smooth border convex dorsally. The cisternae interpeduncularis and pontis filled normally.

A right lateral and frontal cerebral angiogram made two days later disclosed a complete block of the anterior cerebral artery at its junction with the anterior communicating artery on that side. The middle cerebral vessels filled normally and were not displaced. The ophthalmic artery filled unusually well and could be followed well into the orbit.

The patient developed a transient hemiparesis after the cerebral angiogram, but recovered gradually. It was decided not to operate at that time.

Case III. J. M., a man, aged forty, had had double vision for about four months. Photophobia had been present for about five years, and he was accustomed to wearing tinted glasses. There were no scotomas. He became dizzy when bending down or doing heavy manual work. He had had a left frontal headache for about one month before admission, and three or four episodes of projectile vomiting occurred in the three months before admission.

Physical examination revealed normal ocular fundi. The patient stated that he could produce double vision when looking toward the right, upward and downward. The pupils reacted to light and accommodation.

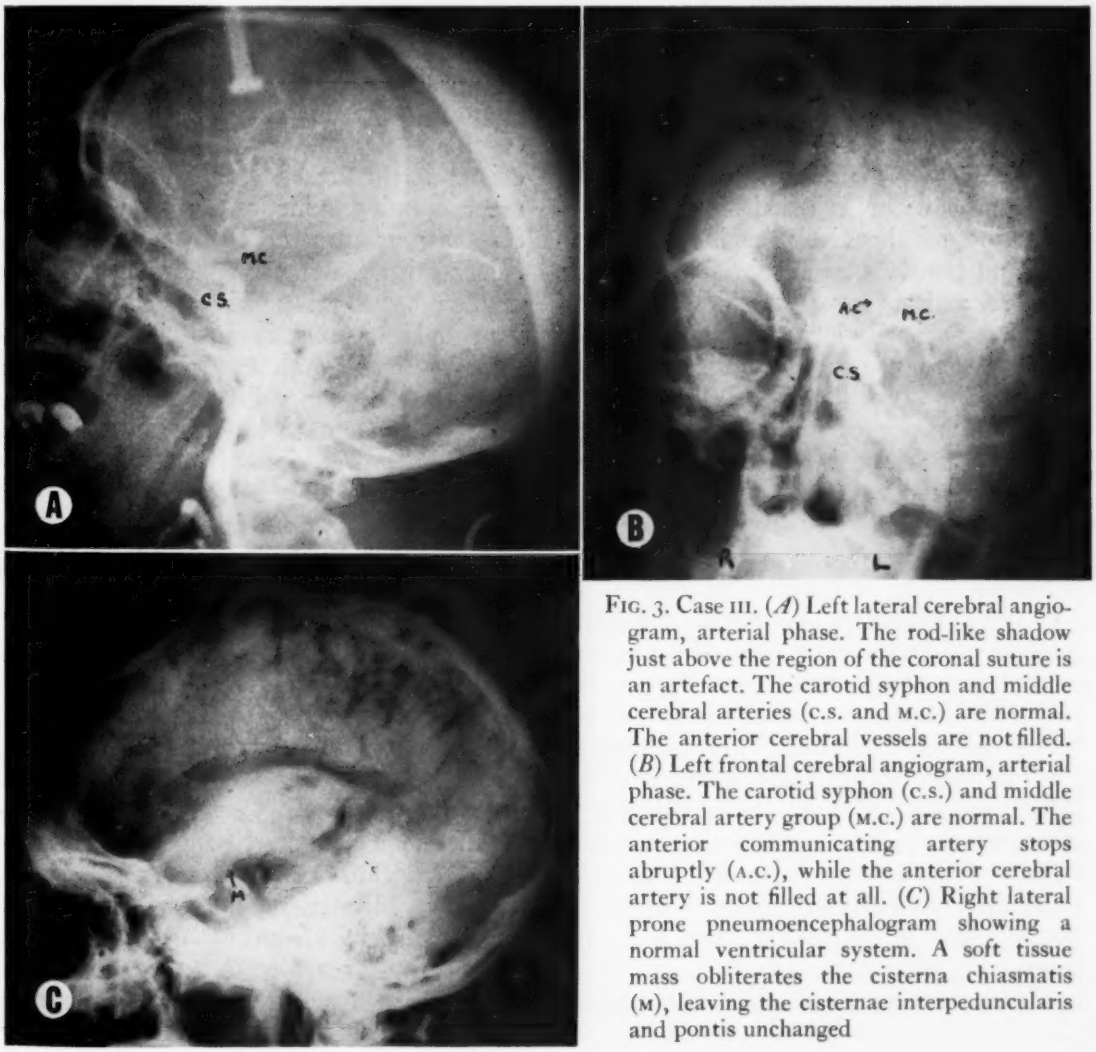

Fig. 3. Case III. (*A*) Left lateral cerebral angiogram, arterial phase. The rod-like shadow just above the region of the coronal suture is an artefact. The carotid syphon and middle cerebral arteries (c.s. and M.C.) are normal. The anterior cerebral vessels are not filled. (*B*) Left frontal cerebral angiogram, arterial phase. The carotid syphon (c.s.) and middle cerebral artery group (M.C.) are normal. The anterior communicating artery stops abruptly (A.C.), while the anterior cerebral artery is not filled at all. (*C*) Right lateral prone pneumoencephalogram showing a normal ventricular system. A soft tissue mass obliterates the cisterna chiasmatis (M), leaving the cisternae interpeduncularis and pontis unchanged

Plain roentgenograms of the skull were normal. Pneumoencephalographic examination showed the ventricular system to be normal in size, position and configuration. The cisterna chiasmatis was obliterated by a soft tissue shadow with a dorsal convexity. The cisternae interpeduncularis and pontis were normal. Left lateral and frontal cerebral angiograms disclosed complete block at the junction of the anterior communicating and anterior cerebral arteries.

COMMENT

Plain roentgenographic examination of the skull is usually of little help in the diagnosis of intracranial aneurysm unless the sac is sufficiently large to produce pressure effects on the adjacent sella turcica, optic foramen or sphenoid bone. Infrequently the wall of the aneurysm may be impregnated sufficiently with calcium to be visible on the roentgenograms. Pneumoencephalographic examination may disclose the shadow of a mass in the vicinity of the optic chiasm protruding into the cisterna chiasmatis without disturbing the configuration of the cisternae interpeduncularis and pontis. Changes in the ventricles are rare, and are not seen unless the aneurysm is sufficiently large to impinge on the foramen of Monro or the inferior surface of the anterior horn. Unless outlined by calcium, the mass protruding into the basilar cisterns cannot be identified as an aneurysm pneumoencephalographically. This sign can only be regarded as nonspecific, one which may be observed with a variety of lesions in the suprasellar region.

Cerebral angiography is the most reliable method of diagnosis for the detection of intracranial aneurysms as well as other pathologic changes in the circulation of the brain and its coverings. The pooling of contrast medium, preferably diodrast, within the aneurysmal sac not only definitely identifies the nature of the lesion, but also affords very accurate information as to its exact site. The diodrast usually lingers in the sac for an appreciable time after the arterial circulation has emptied.

The occurrence of intra-aneurysmal thrombosis which might prevent the entrance of contrast medium seldom has been mentioned. However, the present 3 cases, particularly the first in which necropsy confirmation was obtained, indicates that such blockage is a definite possibility. The classical clinical syndrome of intracranial aneurysms in the vicinity of the cavernous sinus and the circle of Willis consists of headache, sometimes with signs of subarachnoid bleeding together with neurologic symptoms referable to the second, third, fourth, fifth or sixth cranial nerves individually or in varying combinations depending on the location and size of the aneurysm. The necessity for roentgenologic confirmation of the diagnosis requires the judicious use of angiographic and pneumoencephalographic techniques after plain film examination. If the cerebral angiograms show the aneurysmal sac, little else is required. However, if there is a block of the anterior cerebral artery, such as existed in the present 3 cases, further pneumoencephalographic investigation may disclose a mass protruding into the cisterna chiasmatis, leaving the cisternae interpeduncularis and pontis unchanged. The ventricular system in such instances will probably be normal, and it is very easy to overlook the change in the cisterna chiasmatis. The error to be guarded against is the misinterpretation of such pneumoencephalographic studies as within normal limits. If this is done it is only logical to assume that the block at the origin of the anterior cerebral artery may be considered indicative of thrombosis, which is only a partial truth if the coexistence of an aneurysm is overlooked. The clinical management of the patient may also be considerably influenced by accurate diagnosis in such cases.

In the first case reported here, the pneumoencephalographic changes were of great interest because of the seepage of gas into the left subfrontal region. This was due to disruption of the frontal lobe by infarction, and necropsy revealed that the gas escaped alongside a large clot in the cerebrum

which pierced the frontal horn. As pointed out by Elvidge and Feindel,[1] pneumoencephalography might be misleading because retained hemorrhage may extend for some distance from the actual source of the bleeding. This possibility might be compensated for by making sure that the pneumoencephalograms portray the basilar cisterns as fully as possible, and that there is no mass in the vicinity of the cisterna chiasmatis.

SUMMARY

A verified case of thrombus plugging an aneurysm at the junction of the anterior communicating and anterior cerebral artery is reported, together with two additional cases presenting very similar clinical syndromes and roentgenologic changes. Plain roentgenograms of the skull were normal. Cerebral angiography revealed abrupt blockage at the junction of the anterior communicating and anterior cerebral arteries, preventing the diodrast from entering the anterior cerebral arteries while the middle cerebral group filled normally. Pneumoencephalograms revealed a smooth mass bulging into the cisterna chiasmatis, leaving the cisternae interpeduncularis and pontis unchanged.

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THE ROENTGEN LOCALIZATION OF THE BRONCHO-PULMONARY SEGMENTS BY MEANS OF LAMINAGRAPHY, PARTICULARLY IN LUNG TUBERCULOSIS

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THE need of exact topographical analysis of pathological changes in the lungs has increased significantly in the last few years. The surgical management of localized lung tuberculosis experienced a marked impetus with the ability of the internists to influence effectively the tuberculous disease by antibiotics. Help must be given to our chest surgeons in accurate anatomical localization, and for this purpose laminagraphy (tomography) has increased in importance.

If we want to come closer to our ideal goal of delineating pathology as far and as exactly as possible, we must try to get the small nodules in the laminagraphic plane, to force the center of the spherical structures into this plane, and to get the cannular structures along their axis. This requires the improvement of our methods in several directions. In previous periods of development of phthisiology, we had to decide if there were any pathological changes and if evidence of destruction existed. Laminagrams of both lungs in the anteroposterior direction in 1 to 2 centimeter distances were satisfactory for this purpose. Today the development of surgical therapy, particularly the "small resections," demands a higher degree of accuracy.

In the technique of laminagraphy, we particularly appreciate good contrast in the films. We try to hit the pathological changes, possibly with the central ray. And for this reason, we prefer to make laminagrams of each lung separately. We know that the zone of the inmost shadow, in the ordinary excursion angle of 40 to 50 degrees, amounts to 0.25 to 0.5 cm. Therefore, exposures are made of the region concerned in distances of 0.5 cm. Certainly it is true that in equal angles of tube excursion, the planigraphic unsharpness and blurring-out decreases by increasing the focus-film distance. However, in conformity with Bronkhorst, we prefer the greater focus-film distance. The roentgenograms gain in sharpness and clearness, distortion and enlargement being avoided, with the patient in horizontal position at a distance of 140 cm.

The simple survey chest roentgenogram teaches us that only a combination of posteroanterior and lateral exposures gives an idea about the localization of pathology in space as well as its size and shape. The same thing is true, even in higher degree, for laminagraphy. Lateral laminagraphy has so far not been generally performed because of technical reasons. The consideration that the normal hemithorax in the anteroposterior direction in a grown person is about 20 to 25 cm. deep, and in the lateral direction only 13 to 16 cm. wide, shows the advantages of a lateral view laminagram. The technical difficulty of penetrating the thickness of the thorax in the lateral direction does not present any problem with the new apparatus and tubes. As a rule, it is enough to increase the kilovoltage by 10 kv., and to decrease the focus-film distance from 140 cm. to 130 to 120 cm. in thick persons in order to get good roentgenograms. The fact that the normal hemithorax in the anteroposterior direction has a larger diameter than in the lateral suggests that the lateral laminagram presents a larger area projected with a greater probability of bringing out the

pathological changes in comparison with the common anteroposterior laminagram. In lateral position, with the patient on his affected side, layers 4 to 11 cm. are usually sufficient in order to get the whole hemithorax from the lateral chest wall to the hilus. The lateral laminagrams offer a clearer survey than the anteroposterior views, giving a more accurate anatomical localization, particularly where differentiation of single lobes is concerned. In comparison with the simple lateral survey film, the lateral laminagram has the advantage of avoiding the interference of shadows from both sides. The more experience we have with this technique, the more we appreciate the importance of the lateral laminagram which offers us the most valuable evidence, particularly concerning the condition of the lower lobe.

Definite progress has been made in topographical analysis of pathological changes in the lungs by doing routine combination of anteroposterior and lateral laminagraphy. Up to this time, phthisiologists have shown relatively little interest in anatomy of the lungs. Progress achieved in this field has come chiefly from anatomists, radiologists, surgeons, and laryngologists. Most occasions for accurate studies of the topographical anatomy of the lungs have been offered to clinicians by the localization of tumors and suppurative lung diseases. It is well known today that many inflammatory changes in the lungs, and quite often localized lung tuberculosis, are limited to the anatomical and functional unity of the bronchopulmonary segments. It may be caused by the fact that changes in segmental bronchi play an important part in the pathogenesis of these diseases. Of special importance for the development of our knowledge about the bronchopulmonary segments is the examination made on lungs of corpses with casting of the bronchi and the rich experience achieved through bronchography performed on patients. The accepted nomenclature is based mostly on suggestions made by Jackson and Huber in 1943. The International Congress of Oto-Rhino-Laryngology, held in London in 1949, adopted a nomenclature which is only slightly different from that of Jackson and Huber. This nomenclature was also accepted by the meeting of the Thoracic Society on February 24, 1950, as the official nomenclature. It was also agreed that each bronchopulmonary segment should receive a standard number.

The knowledge of bronchopulmonary segments must be considered today as the basis of our anatomical orientation of the lungs. Bronchography is the standard method used on living men. However, in lung tuberculosis, particularly in active forms, bronchography should not be used routinely, as is also emphasized by Huizinga and Smelt. The possibility of the damage done, both by anesthesia and the contrast material, precludes the extensive use of this diagnostic method in lung tuberculosis. Our experience with combined anteroposterior and lateral laminagrams has taught us that in most cases we are able to localize the pathological process by this simple and safe method. Our diagnostic results have been tested in some cases by our chest surgeon, Dr. Pauzner, when performing resection therapy, particularly lobectomy and segmentectomy. Our results have been tabulated in the accompanying chart (Fig. 1), in which we have noted rough mapping of single segments. The distance in centimeters has been calculated in the anteroposterior laminagrams in horizontal position from the back, and in lateral laminagrams from the lateral chest wall of the affected side facing the film.

Usually, the orientation of segments by the accompanying chart (Fig. 1) is sufficient, but there are always cases where accurate localization is rendered more difficult because of borderline situated changes, especially in cases of shrinking and retraction. Naturally, in these cases bronchography might be used for making a decision. In the following, the single segments are briefly described according to

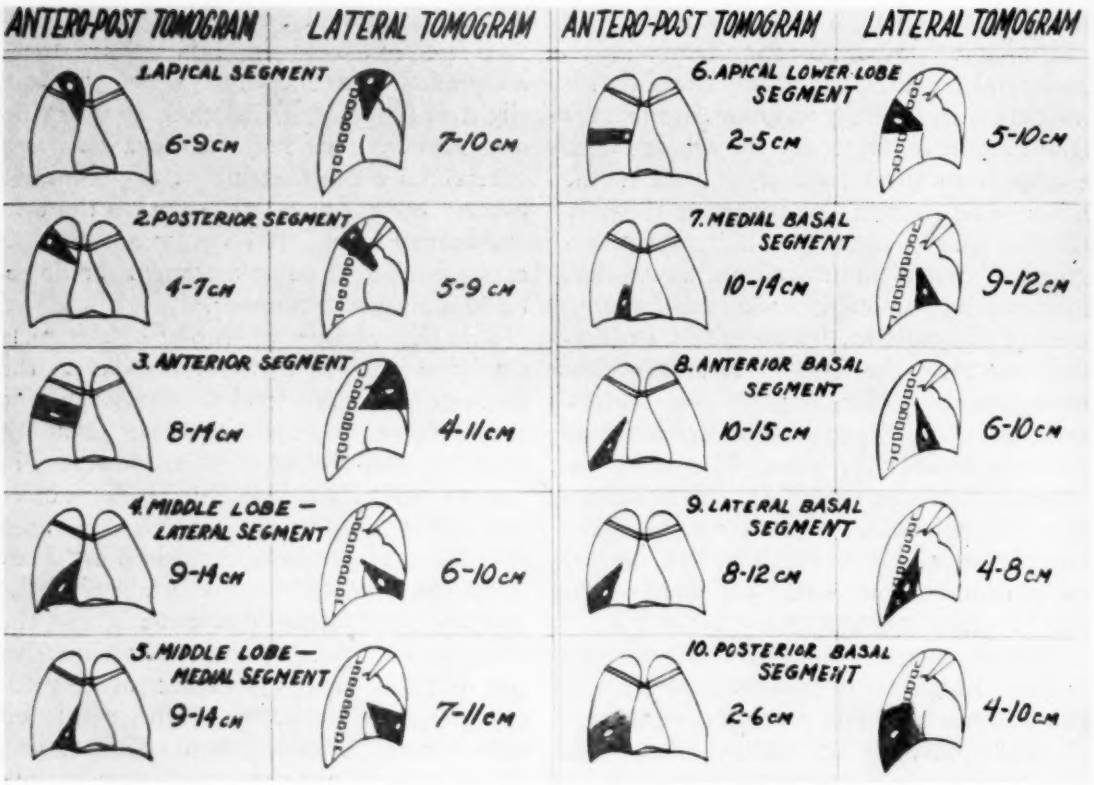

Fig. 1. Distribution of bronchopulmonary segments according to laminagraphic layers.

our own experience with use of the classical descriptions of Brock, Huizinga and Smelt, Temple and Evans, and others.

1. *Apical Segment.* This is supplied by the medially placed ascending apical bronchus which divides into anterior and posterior branches. This segment extends medially to the mediastinum, downward to the superior margin of the hilus, laterally almost to the lateral chest wall at the level of the first rib, upward to the dome of the apex. This segment is projected in the anteroposterior laminagram in the medial portions of the upper lobe, usually in layers 6 to 9 cm. from the posterior chest wall. In lateral laminagrams it extends, usually in layers, 7 to 10 cm. from the lateral chest wall, upward as a wedge, with its top at the hilus. In thick persons it is sometimes hard to visualize in this projection because of large diameters in the shoulder girdle.

2. *Posterior Segment.* The bronchus divides into lateral and posterior branches. In the anteroposterior laminagram this segment includes the posterior portions of the upper lobe in direct connection with the posterior ribs, approximately in layers 4 to 7 cm. Usually, it lies farther laterally than the apical segment and does not reach the mediastinum in the medial direction. It is easier to present this segment in the lateral laminagram than in the apical one. Its lower border runs in the lateral view in the interlobar fissure from the hilus obliquely backward and upward, usually seen in layers 5 to 9 cm. from the lateral chest wall. In this projection, the posterior segment includes the portions of the upper lobe lying upward, backward, and more laterally. Shrinkage and retraction may sometimes cause dislocation of the apical and the posterior segment. These two segments have an important part in the loca-

tion of destructive lung tuberculosis.

3. *Anterior Segment.* The bronchus divides into an anterior and a lateral axillary branch and supplies the anterior and axillary portions of the upper lobe. In the anteroposterior projection, this segment looks nearly rectangular and includes in laminagrams the layers 8 to 14 cm. from the hilus to the lateral chest wall, upward to the first costochondral junction, downward to the fourth anterior rib. We gain a better insight into this segment on the lateral laminagram because we see its whole depth from the hilus to the anterior chest wall, usually in the layers 4 to 11 cm. Its lower border on the right side is shaped by the transverse fissure. Shadows belonging to the anterior branch of the anterior bronchus are better presented in the lateral tomogram.

4 and 5. *Middle Lobe, Lateral and Medial Segment.* The bronchus arises from the anterior wall of the stem bronchus and runs mostly in an anterior, downward direction. About 1 to 2 cm. from its origin it divides into the two main branches. The angle between the branches amounts to about 15 to 20 degrees, the medial bronchus being the upper branch in lateral laminagrams. Lobar involvement, where there is no shrinkage, occupies almost the entire anterior lower part of the right lung field. In the lateral view, the lobe is easily recognized by its wedge shape, with the top on the hilus and the base on the lower part of the sternum. Its position between both interlobar fissures is particularly clearly distinguished in lateral laminagrams.

In anteroposterior laminagrams the location of the lateral segment is more lateral and posterior than the medial segment. The latter includes the anterior and medial portions of the middle lobe and forms, in case of involvement, a quadrangular shadow in connection with the right border of the heart. Both segments are found in the anteroposterior laminagrams in layers from 9 to 14 cm., somewhat farther forward in thick persons. In lateral laminagrams the lateral segment begins somewhat earlier, in layers 6 to 7 cm. from the lateral chest wall, the medial segment usually in layers 7 to 8 cm.

6. *Apical Lower Lobe Segment.* The segmental bronchus arises from the main bronchus opposite to the middle lobe bronchus in the dorsal direction. It divides into three branches: one upper oblique, one lower oblique with medial direction, and one lower oblique with lateral direction. The segment has a pyramidal shape and occupies the dorsal upper part of the lower lobe. Laterally it reaches the posterior portions of the axilla. This segment has, just as the posterior and apical segment of the upper lobe, a special significance in the pathology of inflammatory diseases of the lung, particularly in tuberculosis. Because of its horizontal and dorsal course, the segmental bronchus is easily kinked, particularly during pneumothorax treatment, and so causes the formation of tension cavities which, being placed near the pleura, may perforate. The apical lower lobe segment was implicated in 10.6 per cent of the 110 cases of localized destructive lung tuberculosis, which we analyzed closely in relation to their segmental distribution. Sweany has found that among 268 cases of "primary reinfection cavities" 13.7 per cent were located in the apex of the lower lobe. According to our experience, the cavernous processes in this segment seem to be particularly suitable for segmentectomy. Densities in this segment appear in the survey roentgenogram as shadows in the "hilus area." They may extend laterally as circular shadows and reach the mid-lung field. When using imperfect technique, we may fail to find these cavities, especially on the left side, in a simple posteroanterior survey film. In this segment, laminagraphy shows its particular efficiency. We can see the pathological changes in the anteroposterior laminagram at the level of the hilus near the spine, starting from layer 3 cm. and reaching forward to 5 to 6 cm. The cavities located

in this segment show, as a rule, a sharp round shape in this projection. However, the lateral laminagram offers a better insight into the pathological process. In the lateral laminagram, this segment is seen, in layers 5 to 10 cm. from the lateral chest wall, as a triangular shadow extending from the main bronchus backward and upward. The upper border is formed by the posterior part of the oblique interlobar fissure. This presents a good guide by limiting this segment against the adjoining posterior segment. As a rule, we are able to recognize the main segmental bronchus as a fine horizontal band running backward to the posterior chest wall.

7. *Medial Basal (Cardiac) Segment.* The segmental bronchus arises from the right lower lobe bronchus about 1.5 cm. below the apical bonchus of the lower lobe and runs downward anteriorly. This segment has also the shape of a pyramid whose top lies at the origin from the lobar bronchus and the base on the medial border of the diaphragm, thus forming the anterior medial border of the right lower lobe. We have not yet observed any involvement of this segment in lung tuberculosis by means of laminagraphy. According to its location, it should be found in the anterior layers above the diaphragm entirely medially. In the lateral laminagram it is probably partly overlapped by other segments close to it, particularly the anterior basal and the middle lobe.

8. *Anterior Basal Segment.* Its bronchus arises from the anterolateral aspect of the lower lobe bronchus and runs obliquely downward, forward and laterally. In anteroposterior laminagrams, this segment can be distinguished from the other basal lower lobe segments, which it partly overlaps, only by its position in anterior layers. We see wedge shaped and quadrangular shadows above the lateral portions of diaphragm. In the lateral laminagram this segment can be seen in layers 6 to 10 cm. above the diaphragmatic dome. It is in this projection separated from the lateral middle lobe segment, or the inferior lingular segment on the left side, by means of the interlobar fissure. This segment, as the other basal ones, is often the seat of secondary dissemination in lung tuberculosis.

9. *Lateral Basal Segment.* Its bronchus is likewise a ventral branch of the lower lobe bronchus. It runs downward and laterally; and in normal posteroanterior survey films, it overlaps the other basal segments. In anteroposterior laminagrams in middle layers it occupies the lateral portions of the lung above the diaphragm. In the lateral laminagram it partly overlaps the posterior basal segment, but it lies slightly more ventrally and also comes closer to the lateral chest wall. It can be found in the lateral laminagraphic plane in layers 4 to 8 cm.

10. *Posterior Basal Segment.* Its bronchus runs downward, backward and slightly laterally. This segment occupies the lower two-thirds of the dorsal surface of lower lobe, while the upper third is formed by the apical lower lobe segment. Also, the back part of the diaphragmatic surface of the lower lobe belongs to this segment. Because the segmental bronchus presents the terminal continuation of the lower lobe bronchus, this segment is often the seat of aspiration, particularly in lung tuberculosis. In the anteroposterior laminagram the layers 2 to 6 cm. are mostly occupied. The lateral laminagram shows clearer pictures, mostly in layers 4 to 10 cm. The lower lobe cavities are often located in the posterior basal segment. If they are placed sufficiently high in this segment, it may be difficult to distinguish whether they belong to this segment or to the apical lower lobe segment or even to a supernumerary subapical segment.

It is always advisable, before excision therapy in the upper lobe, to take a lateral laminagram of the affected lung in the layers 4 to 11 cm. because this gives the best evidence about the condition of the lower and middle lobes.

As far as segmental division is concerned, the *left side* differs, as is known, from the right, by having one apicoposterior seg-

ment (1 and 2) supplied by a common segmental bronchus arising from the left main bronchus. Both middle lobe segments (5 and 4) correspond to the lingular segments (4 superior and 5 inferior lingular segments). It must be mentioned that the left lower lobe bronchus has no medial basal branch. Consequently, there is no segment on the left corresponding to 7. Therefore, the number of segments on the left is reduced to 8.

The presented laminagraphic schedule and the description of the location of the single bronchopulmonary segments form the frame which allows us in most cases to decide to which segment a pathological process belongs. However, we cannot follow the segmental bronchus along its entire course as we do in bronchography. We may succeed only if we get its longitudinal axis in the laminagraphic plane. That happens in the simple anteroposterior laminagram with those bronchi which run in the frontal plane; for instance, the perpendicularly ascending branch of the apical bronchus and the lateral branch of the anterior bronchus. The other bronchi, after arising from the lobar bronchus, take an oblique direction and may be presented at most by small sections of their course. It should be noted that the segmental bronchi sometimes do not show any straight line course.

In order to get the longitudinal axis of a segmental bronchus into the laminagraphic plane, we have to use oblique positions. Following this consideration we have used this simple method in our work: We have compensated the level differences between the concerned lobar bronchus and the pathology by putting cotton rolls under the patient while he is in the horizontal position, so that the segmental bronchus comes to the horizontal laminagraphic plane. For instance, a cavity of the right upper lobe lies laterally under the clavicle and is, in the anteroposterior laminagram, best seen in the layers 4 to 5 cm. It probably belongs to the posterior segment. The right upper lobe bronchus in the hilar area is best presented in layers 10 to 11 cm. Thus there is a level difference of 6 to 7 cm. between the cavity and the upper lobe bronchus. We raise the posterior chest wall in the probable cavity area up to the bronchus level and then take anteroposterior laminagrams of the concerned area in layers 10 to 14 cm. It corresponds to the right anterior oblique position described by Huizinga and Smelt. In this way we often succeed in presenting laminagraphically the bronchopulmonary segment in the longitudinal axis of the segmental bronchus. We are able thus to obtain a better insight into the pulmonary focus and its bronchovascular connection.

1. *The Pulmonary Focus.* Pulmonary foci which have originated via the bronchial route maintain their connection with the bronchial tree. Isolated cavities present, principally, the typical picture of a tennis racket provided that a correct segment-axial projection has been made. Thus, these cavities present, as a rule, an ovoid shape. They gain either a round or an irregular form if the laminagraphic plane cuts the segmental axis perpendicularly or obliquely. It is our impression that the oval shape is caused by traction through the bronchovascular bundle. Most cavities or tuberculomas appear as round shadows in anteroposterior laminagrams, while in the lateral or oblique laminagram, they appear rather as oval shadows. This behavior is particularly characteristic for cavities in the apical lower lobe segment. It is obvious that the permanent pulling, especially on respiration, presents an important causal factor. On the other hand, other pathologic changes, such as tumors, cysts, scars (processes which have no direct relation to the bronchus) do not show this behavior. It is therefore advisable to look at the shape of the shadow in the lateral or oblique laminagram when making a differential diagnosis between tuberculoma and tumor.

2. *The Segmental Bronchus.* One of the most important tasks of our diagnostic study is to recognize pathological changes in the segmental bronchus. The signifi-

cance of changes in the bronchial wall proximal to the orifice of the cavity is well known. Bronchoscopy permits the visualization only of changes in the larger bronchi whose disease already presents an advanced stage of bronchial tuberculosis. Bronchography, which has proved to be so important in the diagnosis of nonspecific lung diseases, may rather give evidence about the condition of the smaller bronchi. However, the use of bronchography in lung tuberculosis as a routine method is quite limited because of reasons mentioned above. Besides, the iodized oil introduced into the smaller bronchi creates unphysiological conditions and causes functional changes in the bronchial branches. We believe that laminagraphy might offer greater advantages because of its simplicity.

While considering the "broncholaminagram," we were able to establish the following types of pathological changes in the segmental bronchi:

a. Pathological Air Filling: In the course of our observations we have found that, as a rule, there is no air column in a normal segmental bronchus although the laminagram is routinely taken at the top of inspiration. It is well known that the segmental bronchi and their branches run together with the ramifications of the pulmonary artery. These arterial branches are the anatomical basis for the many dichotomical stripes seen on a simple roentgenogram of the lungs and even better on a normal laminagram. They serve as a guide for the concomitant bronchial branches when analyzing the laminagraphic views. It is not yet clear why the aeration in the lumen is limited to the larger bronchi, *i.e.*, the main bronchus, the lobar bronchi and the initial parts of the segmental bronchi. That may be caused by a technical roentgenological reason (small diameter of the air column) or by a physiological one (contraction of the smooth musculature). However, an aerated lumen may be found quite often in the segmental bronchus when we are dealing with pathological changes in its area, and this fact may lead us to the pathology. This finding may be caused by thickening and functional damage of the bronchial wall, its contents of mucus and secretions in the lumen originating from the cavity, and inflammation of the bronchial mucosa. We may explain the open lumen in the same way as in bronchography when the iodized oil opens the lumen of the smaller bronchi, filling it with oil and air alternating. According to Luisada's electrobronchographical investigations, there is evidence that the muscles take an active part in the expansion and recoil of the lung during respiration by rhythmic contraction and relaxation. It is obvious that pathological changes of the smooth muscles along the small bronchi may disturb this rhythmical contraction (Fleischner).

b. Irregularity of the Bronchial Wall: There might be windings and recesses of the bronchial wall, in pathological cases, due to chronic peribronchial shrinking. Sometimes we may succeed in getting into our plane a partial narrowing of the bronchial lumen. Bronchi of this kind have mostly an irregular course and could be visualized in the laminagram only partly, even in correct positioning.

c. The Rosary-Shaped Bronchus: The segmental bronchus sometimes shows regularly distributed condensations of the wall presenting proliferative changes in chronic inflammatory processes.

d. The Distended Bronchus: If the peribronchial space appears as a regular condensation, the bronchus shows a marked airy lumen in laminagrams. One gains the impression of the bronchial wall being distended by traction. We see this kind of appearance in acute exudative processes, and there is good suggestive evidence that this appearance is caused by inflammatory processes of the bronchial wall. However, peribronchial atelectasis may also take part in this distention.

The presentation of bronchial changes in laminagrams is of special value because the thickened bronchial branches filled with air point directly to the pathology.

Of course we cannot decide in this way whether we are dealing with specific or nonspecific changes.

SUMMARY

The routine combination of anteroposterior and lateral laminagraphy gives us a rough picture of the location of pathological changes in the lungs. The distribution of single bronchopulmonary segments is discussed. A scheme is established to determine by laminagraphy to which segment a pathological process belongs. Our judgment must be carefully made in processes located on the border of two segments and in cases of dislocation following shrinking. It is often possible to present laminagraphically the segmental bronchi in their longitudinal axis by adding a suitable oblique position. The pulmonary focus appears then in its characteristic shape, cavities usually ovally shaped because of the traction carried out by the bronchovascular bundle. The laminagraphic appearance of pathological changes of the smaller bronchi are discussed. Aeration of the lumen, thickening and distention of the bronchial wall are the most remarkable signs thus obtained.

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VALUE OF ROENTGEN STUDY OF ETHMOID SINUSES THROUGH THE ORBITAL WINDOW*

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IT HAS long been recognized that the diagnosis of ethmoid sinus disease made by the otolaryngologist is not always verified by the roentgenologist. Believing that the discrepancy resides in roentgen technique and interpretation, we were prompted to restudy the problem in relation to these two factors. In reviewing the literature we found that this was by no means a new subject. The problem of correlative findings by the roentgenologist and the otolaryngologist has been a "thorn" for many years. Furthermore, Rhese's[8] work, in 1911 and Mosher's[6] excellent description of the anatomy of the ethmoid labyrinth in 1912 have been a challenge to devise a better technique of ethmoid investigation.

The part ethmoid involvement plays in diseases of the eye has long been a moot subject. The literature on this subject is too voluminous to enumerate in this paper. A review of these papers left us with the impression that the roentgenologic demonstration of the individual cellular detail of the ethmoid labyrinth had not been satisfactorily accomplished. This has been borne out by Proetz[7] who discusses the difficulty in adequate visualization of the ethmoid cells. Various authors have attempted to overcome this difficulty by utilizing additional procedures: Moore[5] described laminagraphy; stereoscopy has been utilized by Grier.[3] However, neither method lends itself to routine study.

With these facts in mind, and with the ophthalmologic department demanding more sound information concerning its eye problems in relation to ethmoid disease, an anatomical and roentgenological study of the ethmoidal labyrinth was undertaken.

TECHNIQUE

Of all the various techniques advocated for the roentgen demonstration of ethmoid cells, that described by Rhese[8] appeared the most fruitful. In 1911 Rhese succeeded in his objective of demonstrating the unilateral visualization of the ethmoid cells by the oblique projection into the comparatively clear orbital cavity. Rhese's contribution to this particular study apparently has not received the consideration which it rightfully deserves. It is our purpose to demonstrate the unique value of a modification of this special view in the roentgen diagnosis of ethmoid disease.

A special view of the ethmoid cells that could be reproduced with a degree of accuracy initiated the use of some mechanical contrivance. Since the optic foramen suggested such an important landmark in relation to the posterior superior border of the posterior ethmoid cells, an apparatus was devised after the principle of the Camp localizer. Camp and Gianturco[1] had developed an optic foramen localizer by carefully studying the obliquity necessary to permit the central beam to pass through the foramen on the dependent side. By modifying the position of the central beam so that it passes through the tip of the anterior clinoid process of the side adjacent to the cassette we were able to reproduce an ethmoid view which varied only in the slightest degree in different type skulls (Fig. 1 and 2).

The greatest asset to the procedure for taking this view is in its simplicity. The patient is placed on the roentgen table in a prone position and the head placed in a lateral starting position. The ethmoid localizer is then placed over the outer canthus of the eye with the upper line on the localizer set parallel to the ciliary border and extending to the upper lobe of the ear. The inferior line extends perpendicularly to the acanthomeatal line. The end of the tongs are then brought parallel to the central beam by adjusting the position of the head. The central ray is then adjusted to the center of the film. The position as well as the localizer is demonstrated in Figures 3 and 4.

The technical factors are 100 ma. at 0.5 sec., 36 inches focal skin distance, with 59 kv. for an

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18 cm. thickness when measured in the oblique position. The uniformity of technique bilaterally is paramount. In addition, it is well to record the technique utilized in those cases where follow-up studies are to be done, so as to permit accurate comparisons.

ANATOMY OF ETHMOIDAL CELLS

The ethmoidal cells are extensions or evaginations of the nasal mucosa from the middle, superior and first supreme nasal meatuses. Early in life the lateral masses of the ethmoid bone and its appendages are solid structures. Soon, however, the lateral masses become more or less honey-combed by the developing ethmoidal cells. Each cell always communicates with the meatus from which it develops.

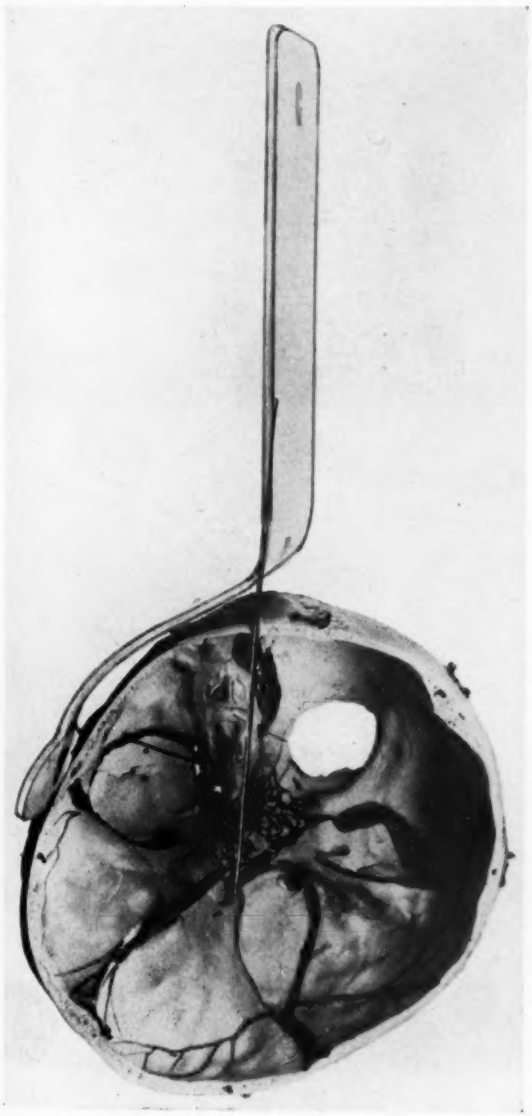

Fig. 1. Section of skull demonstrating the construction of the ethmoid device with the central ray projecting through the anterior clinoid process.

Fig. 2. Positioning of device in lateral projection with central line extending from the canthus to the external meatus.

There is early a topographical division of the ethmoids into two groups: (1) anterior ethmoid cells; (2) posterior ethmoid cells. The anterior group develops from points caudal to the attached border of the middle nasal conchae and the posterior group develops from points superior or cephalic to the attached border of the middle nasal conchae.

During childhood the poserior ethmoid cells gradually pneumatize and make shell-like the superior and supreme conchae. Before puberty the cells may extend into the infra-orbital plate of the maxilla. The discrepancy in the size of the cells at given age is due to the great variability in the number of cells differentiated. This has been demonstrated by Schaeffer[9] in a series that were measured through the childhood period. This author found the number of cells to vary from three to fifteen.

The great complexity of the adult ethmoidal labyrinth and the variations in shape and disposition of the individual cells composing it are in accord with the multipotentialities of development.

The fully developed ethmoidal capsule occupies the field between the medial surface of the bony orbits and extends between the frontal sinuses ventrally and the sphenoidal sinus dor-

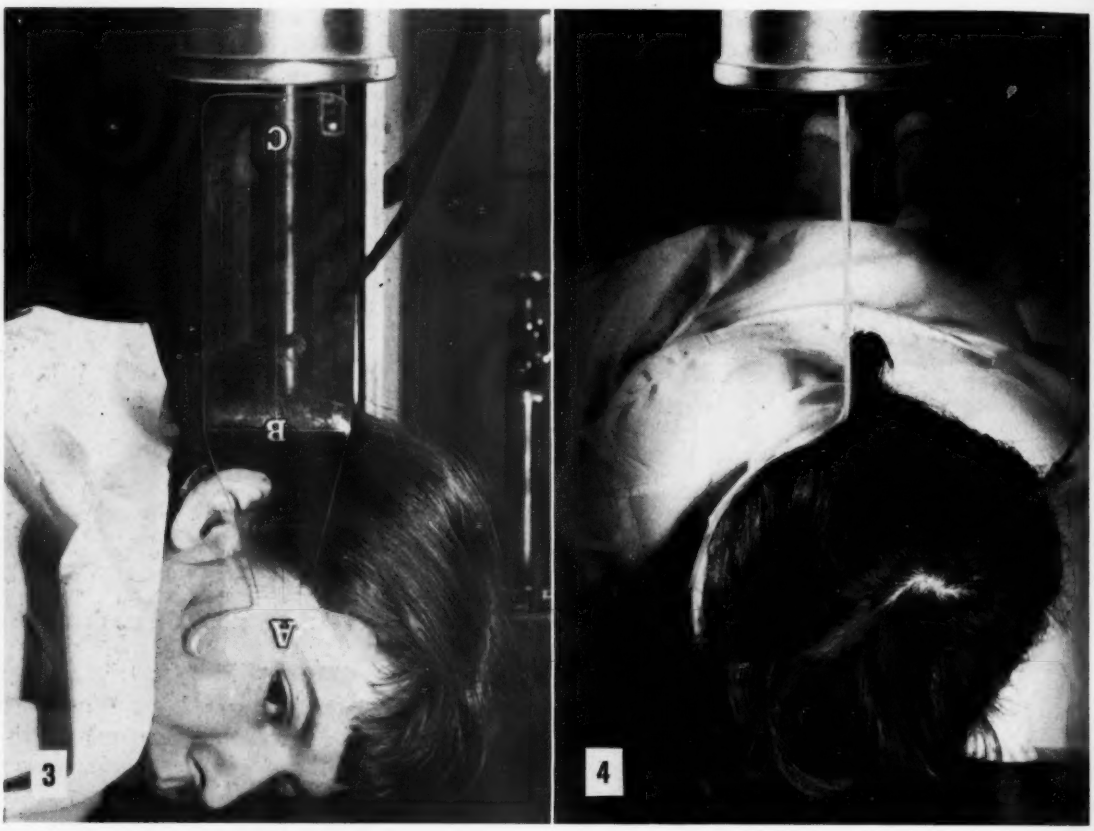

FIG. 3 and 4. Ethmoid localizer applied to human subject.

sally. Above, the cribiform lamina serves as the boundary plane, while the uncinate process represents the caudal limit. However, the ethmoid labyrinth may extend beyond one or more of its above boundaries and thus produce ethmofrontal, ethmosphenoidal, ethmopalatine, ethmomaxillary and ethmolacrimal cells. In addition, cells may extend into the middle nasal concha.

The anterior ethmoid cells vary in number from two to eight. The fewer the number of cells, the larger the volume of the cell. The groups are usually subdivided according to the location of the ostia of the cells concerned. Thus, these cells may be divided into a group communicating with the frontal recess, ethmoid infundibulum, and those whose ostia communicate with the ethmoid bulla. Those cells which expand upward into the floor of the frontal sinus are the most difficult to differentiate on roentgenograms from true frontal sinuses.

The posterior ethmoid cells arise from the superior and supreme nasal meatuses and thus communicate with the nasal fossa above the attached border of the middle nasal conchae. Not infrequently the posterior and anterior group of ethmoidal cells overlap each other topographically, but their natural paths of drainage are preserved.

According to Schaeffer[9] there is no constancy in the number of posterior cells and the number may vary from one to seven.

In the more usual and moderate sized posterior ethmoidal cells the optic nerve at the optic foramen is separated from the most dorsal and superior of the cells by bone from 2 to 5 mm. in thickness. The optic nerve then diverges more and more from the posterior ethmoid cells as one passes from the optic foramen to the eyeball. Some of the posterior cells may extend back into the sphenoid bone or pneumatize cells which extend into the body or lesser wing of the sphenoid and may surround the optic nerve.

DISCUSSION

In our study, a dried skull was used in which the orbital lamina papyracea was

carefully removed so as to expose the cells of the entire ethmoid labyrinth. Various projections were made until the cell structure was best visualized and secondly that only the side adjacent to the cassette was demonstrated.

Figure 5 represents the negative which we feel most clearly demonstrates the cellular outline. We have observed that when the optic foramen and anterior clinoid process of the roentgenographed side form the posterior boundary of the orbit, the majority of unilateral cells are visualized. In addition, they are reproducible and easier to interpret.

To prove that this oblique projection would demonstrate only the unilateral cells, we attached lead numbers to the areas concerned. Number "1" (two of them) was glued to the superior and inferior border of the lamina papyracea of the orbit adjacent the cassette. Number "3" was placed in the posterior portion of the opposite orbit along its medial aspect (on the opposite lamina papyracea). Number "8" was attached to the anterior clinoid process of the opposite side. Letter "L" indicates the clinoid process of the dependent side and lies superior to the optic foramen. Numbers "5" and "6", which are somewhat distorted in the roentgenogram, are attached to the dependent and opposite zygomatic process, respectively (Fig. 6).

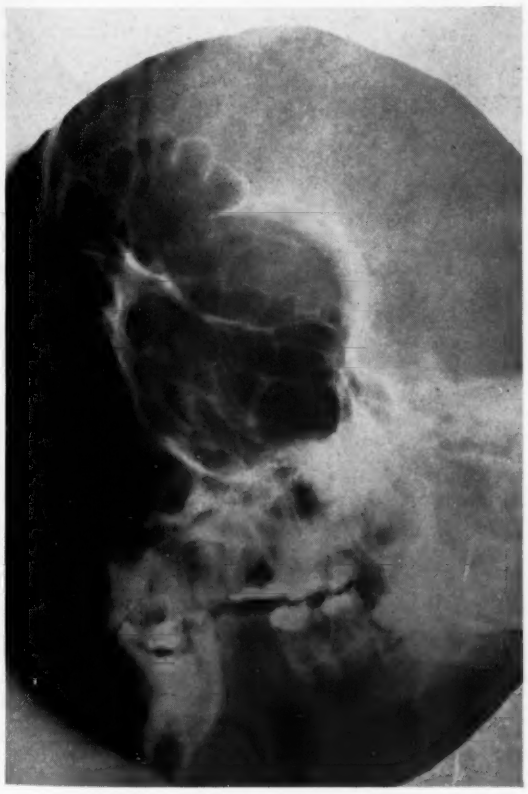

Fig. 5. Reproducible view of "orbital window" with optic foramen lying in posterior portion of orbit. Frontal cells blend with ethmoids.

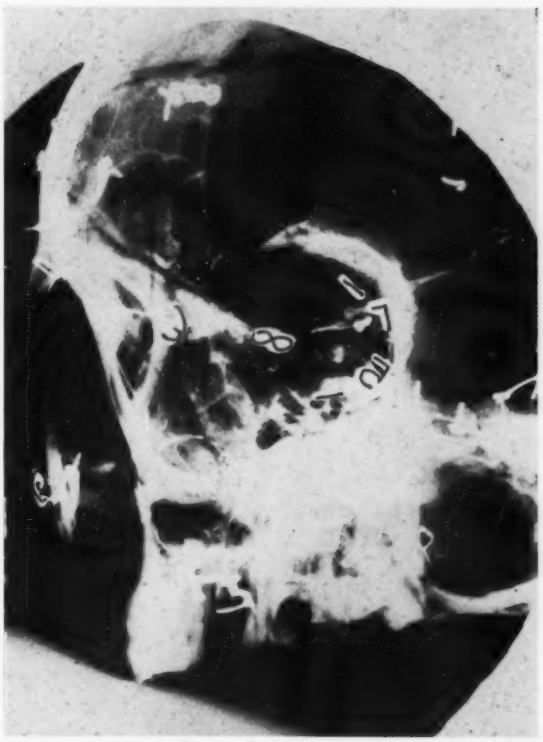

Fig. 6. Study of skull for roentgen distortion was attempted by numbering the important landmarks: No. 1—Superior and inferior border of lamina papyracea. No. 3—Posterior ethmoid cells of opposite ethmoid labyrinth. L—Anterior clinoid process of dependent side. No. 8—Clinoid process of side away from film. No. 5—Zygomatic arch of opposite side. No. 6—Zygomatic arch of opposite side. Note that the posterior ethmoid of opposite side No. 3 is thrown free from window of unilateral cells.

This study aided both in an understanding of the distorted roentgen anatomy as well as providing proof that the opposite orbit and ethmoid cells are thrown anteri-

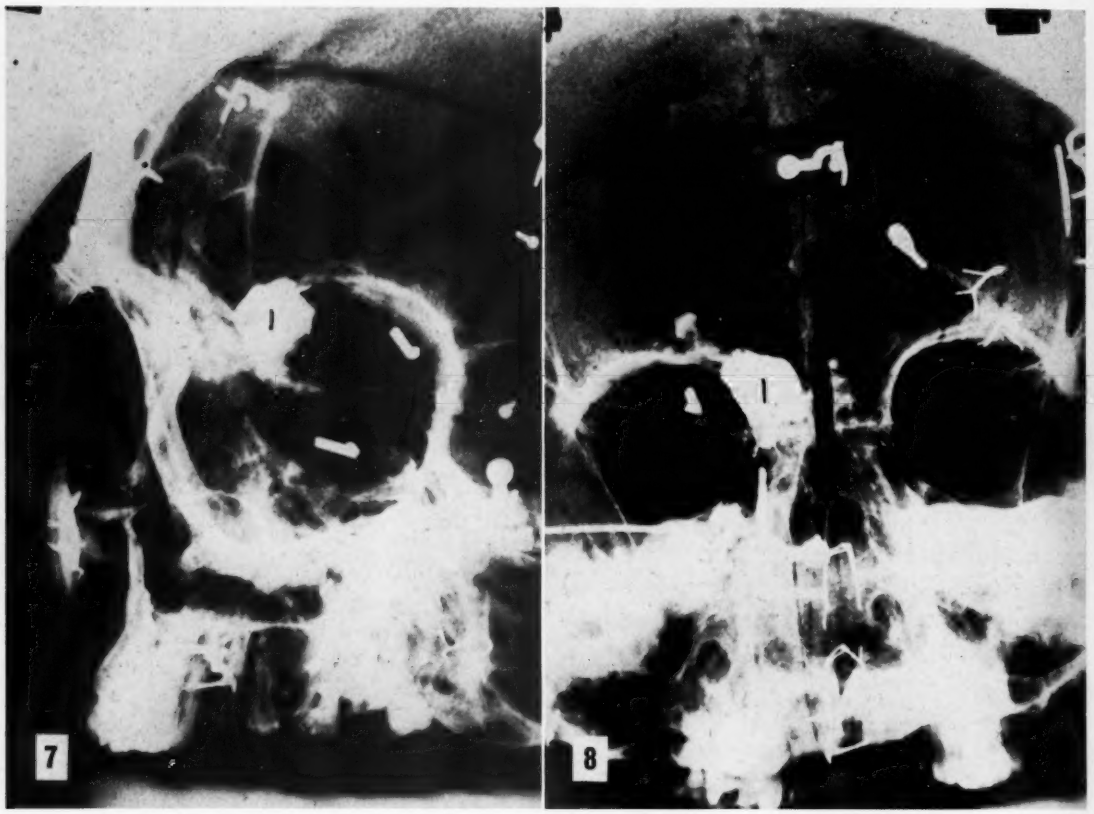

Fig. 7 and 8. Lipiodol filled anterior cell in oblique and posteroanterior projections. The horizontal No. 1 limits the superior and inferior surface of medial aspect of lamina.

orly and therefore not superimposed on the side being investigated. The most posterior ethmoid cells of the opposite orbit are shown to be in the posterior rim of the dependent orbit, as outlined by the Number "3" marker.

The ethmoid cells were filled individually. Cotton which had been soaked in lipiodol was inserted into the cells and a lateral, posteroanterior, as well as an oblique view, was made until the entire labyrinth had been filled.

For reasons of clarity, we have numbered the cells as they were filled so as to allow comparison of their positions in the various projections. Cell No. 1 (Fig. 7) represents an ethmoid recess which, when projected in the posteroanterior view (Fig. 8), is observed as an opacity lying medial and superior to the orbit and extending upward into the base of the frontal sinus on the same side. Cell No. 2 was then filled and the various views are shown (Fig. 9, 10 and 11). Cell No. 2 is a posterior cell which formed the anterior border of the sphenoid sinus and the inferior aspect of the optic foramen.

In the routine posteroanterior projection the clouding is now observed to extend downward from that seen in Figure 8. The posterior cells are projected in such a manner that the opacity extends from the inferior medial aspect of the orbit, medially, and ends just several millimeters from the nasal septum. In the lateral projection, the contrast medium is seen to lie in such position that the individual cells are somewhat obscured by the bones forming both orbits as well as by the frontal processes of the zygomas. Cell No. 2 is thrown just inferior to the lesser wing of the sphenoid and blends with the bone density forming the posterior

FIG. 9, 10 and 11. Demonstrating the value of the orbital view with the lateral and posteroanterior projection. Cell 1 is anterior cell. Cell 2 is most posterior cell.

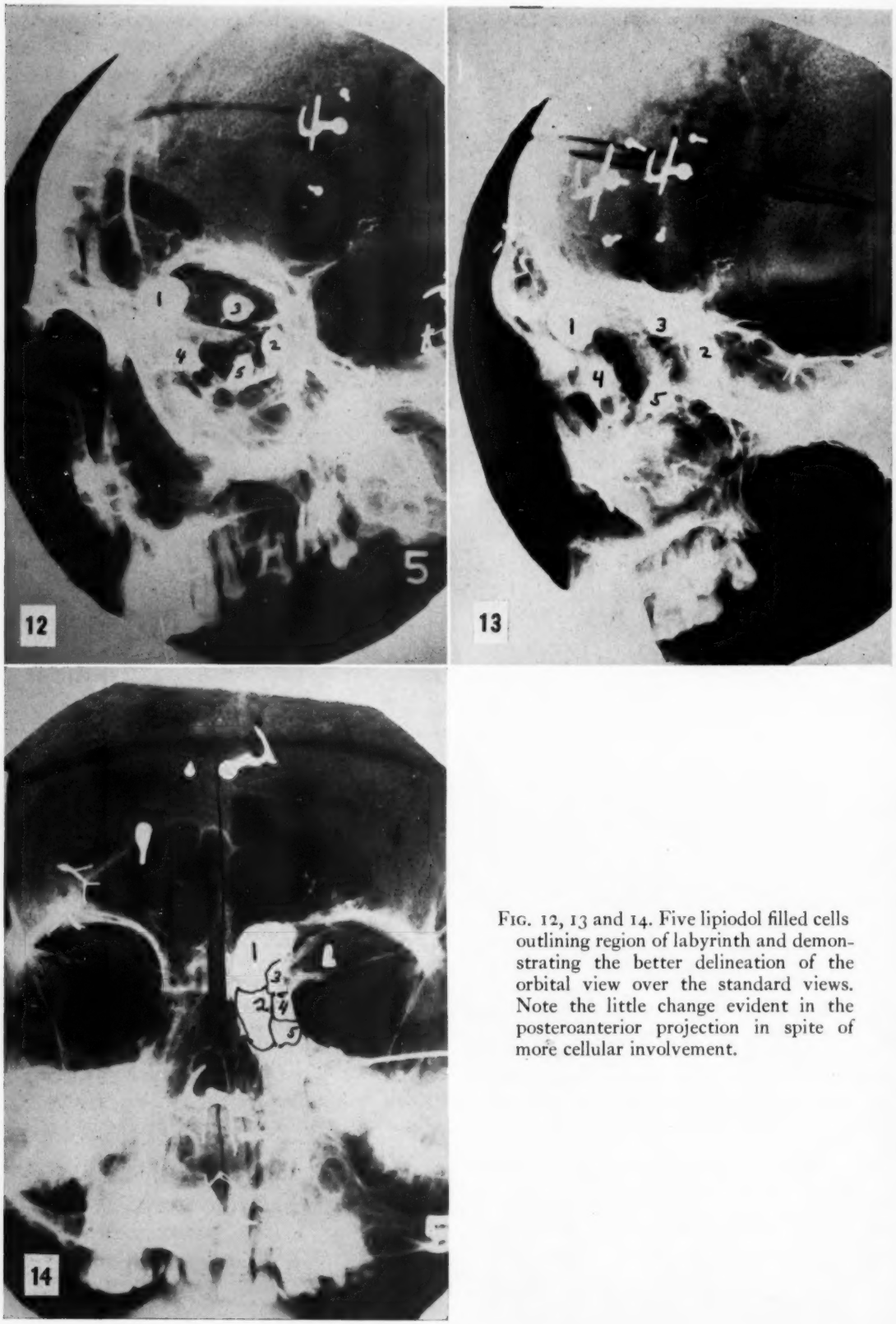

FIG. 12, 13 and 14. Five lipiodol filled cells outlining region of labyrinth and demonstrating the better delineation of the orbital view over the standard views. Note the little change evident in the posteroanterior projection in spite of more cellular involvement.

orbital wall. It is noted that the cellular structure of the remaining ethmoidal cells are not well defined. With subsequent filling of the ethmoids, it became apparent that little information could be gained by the posteroanterior projection. This is demonstrated in Figures 12, 13 and 14, where five cells are filled with only slight significant change in the roentgen appearance of the posteroanterior view alone. Again, the oblique and the lateral views are compared for visualization of the opaque cells. No cell structure could be seen in the latter and the cells are seen to form the orbital outline, being ill defined due to overlying bony structure. It can be readily appreciated that from this lateral roentgenogram one would be unable to determine the position of these cells in regard to whether the right or left ethmoid cells were affected. The oblique view clearly demonstrated the position of individual cells as well as thin interlacing bony structure separating them. With complete filling of the ethmoids, there was little gained by the oblique view as compared to the lateral. However, this would not hold for comparing unilateral involvement. If cells were involved bilaterally, the projection would give no detailed information.

These observations are not new and were well demonstrated in the monograph by Rhese.[8] The diagram (Fig. 15) taken from his original article reveals the anatomical position of the ethmoid labyrinth on the horizontal view. By correlating the various letters in the two sketches, one can readily observe the difficulty in detecting any detailed information from the posteroanterior position (Fig. 14). In segment *a*, the posterior ethmoid cells being located at the margins of the orbit and lying lateral to the anterior and middle groups, are not superimposed on them or the sphenoid sinus in

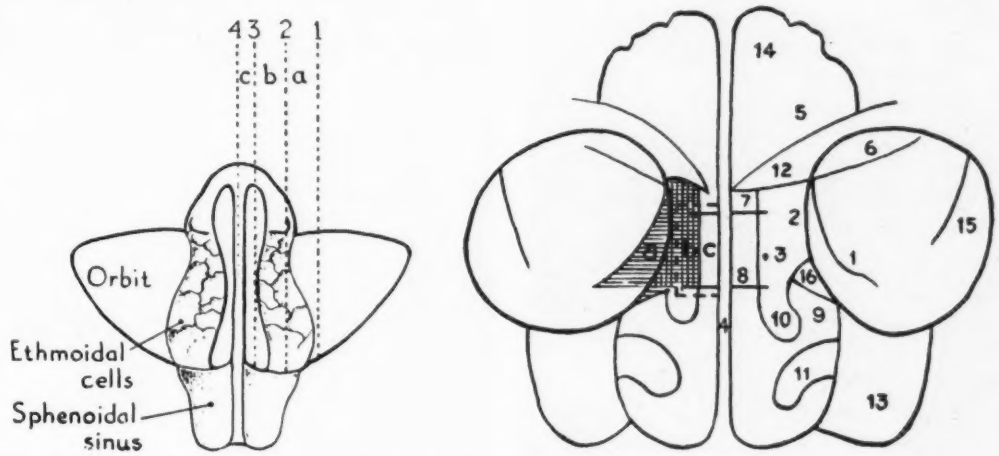

Fig. 15. Relations of the ethmoid labyrinths, sphenoid sinuses and orbits, as viewed in horizontal section. In segment *a*, the posterior ethmoid cells are located on the margins of the orbit lying lateral to the anterior and middle groups and so are not superimposed on them or the sphenoid sinus in the dorsoventral roentgen projection. In segment *b*, the three groups of ethmoid cells lie in the same dorsoventral axis and are, therefore, superimposed on one another and on the sphenoid sinus in the dorsoventral roentgen projection. In segment *c*, a portion of the anterior wall of the sphenoid sinus lies medial to the ethmoid labyrinth and is not superimposed on it in dorsoventral roentgen projection. (Redrawn from Koehler.)

The paranasal sinuses in dorsoventral projection. *a*, posterior ethmoid cells; *b*, superimposed anterior, middle and posterior ethmoid cells; *c*, sphenoid sinus without superimposed ethmoid cells. *1*, inner edge of the inferior orbital fissure; *2*, inner margin of the orbit; *3*, medial border of the lateral mass of the ethmoid bone; *4*, nasal septum; *5*, superciliary ridge; *6*, upper border of the superior orbital fissure; *7*, nasofrontal and nasomaxillary sutures; *8*, roof of the superior nasal meatus; *9*, uncinate process; *10*, middle turbinate; *11*, inferior turbinate; *12*, internal projection of the superior orbital margin; *13*, maxillary sinus; *14*, frontal sinus; *15*, sphenozygomatic suture; *16*, ethmoid bulla. (Redrawn from Koehler.)

the routine posteroanterior projection. In segment *b*, however, the ethmoid cells lie in the same dorsoventral axis; they are therefore superimposed on one another as well as on the sphenoid sinus. In segment *c*, a portion of the anterior wall of the sphenoid sinus lies medial to the ethmoid labyrinth and is not superimposed in the posteroanterior roentgenogram. In the presence of sphenoid involvement, clouding will be demonstrated in an area confined to the interrupted lines. These diagrams which are purely schematic do not accurately reflect the roentgen appearance in all cases. Variations in the ethmoid structure will account for slight roentgen alterations; however, in the main, the fundamental difficulties of making any study of the cells from the posteroanterior and lateral positions is universally accepted.

ROENTGEN INTERPRETATION OF ETHMOID DISEASE

The varying size, location, and number of ethmoid cells has made the roentgen interpretation exceedingly difficult. Law[4] has called attention to the variation of the anterior border of the ethmoid capsule which may possess wide limits. In addition, his discussion on the four appearances relative to the ethmoid septa is well recognized.

While Law described the changes in regard to the lateral view, we believe it necessary to gain some understanding of the roentgen changes with the oblique view.

Again utilizing the dried skull, fully cognizant of the lack of cellular membrane lining these cells, we attempted to gain some understanding of the changes which might occur in ethmoiditis.

FIG. 16. Demonstrates haziness in the acacia filled cells as compared to the central nonfilled ethmoid cells. The ethmoid septa are visible.

FIG. 17. Further concentration of the acacia in the cells causes smudging or loss of the septum due to loss of contrast. Compare with central nonfilled cells.

The cells were filled individually with dried cotton and a roentgenogram made to note any change. No significant change being seen, the cotton was injected with an acacia mixture. The acacia was prepared to a consistency not unlike a purulent exudate. The amount instilled into the cotton was gradually increased until all of the cells were filled. Between each gradation, the oblique view was made with an attempt to determine changes in density of the cells as well as any septal alteration. Slight changes would be difficult to observe on reproductions; however, it was possible in the original roentgenograms to note increasing haziness about the ethmoid cells and the septa becoming somewhat indistinct and smeared. This can be seen in Figures 16 and 17 where the middle ethmoid cells were left uninjected to allow some direct means of comparison.

These findings were again reproduced by injecting a starch solution in increasing amounts into the cotton filled cells.

Thus, without the benefit of the normal mucous lining, we could observe septal changes and clouding of the ethmoid cells. Although the septa were smeared when the cells were most dense, still we were unable to completely obliterate the fine cell wall. These findings are somewhat substantiated in a recent roentgen-ray microspectrographic investigation by Engström, Hamberger and Welin.[2] They concluded that when the cell wall in the ethmoid labyrinth was less than 3 millimeters thick, contrast was lost due to inflammatory exudate filling the cells and not from any destructive phase of the bone surrounding these structures. These findings are verified in the subsequent return to normal of the septal outline with regression of the inflammatory process.

By this artificial method of investigation we were able to reproduce alterations in the ethmoid cells that most likely prevail in disease. In chronic ethmoiditis the changes described by Law[4]—condensing osteitis of the bone structure about the ethmoid cells as well as rarefying osteitis—is well appreciated by the authors and both have been observed clinically.

CONCLUSIONS

Approximately forty-five cases of suspected ethmoid disease have been investigated by an oblique method of roentgenography in which roentgen changes were correlated with clinical observations.

This view has had its greatest value in those cases where:

(1) Clinical evaluation led one to suspect ethmoid involvement but routine views were reported as negative.

(2) Findings on routine views were reported as showing complete opacity of the ethmoid labyrinth, but on further study with the orbital window view only a few cell groups were seen to be involved.

This is especially significant in certain eye conditions where it is important to know whether or not cells in close proximity to the optic nerve are involved, and in cases of neoplasm of the ethmoid where it is important to know how much of the labyrinth has been destroyed.

SUMMARY

1. A special view of the ethmoid cells that could be reproduced with a degree of accuracy, initiated the use of some mechanical apparatus.

2. It was observed that when the optic foramen and anterior clinoid process of the roentgenographed side formed the posterior border of the orbit, the majority of unilateral cells are visualized.

3. The ethmoid cells were studied by an artificial method of investigation simulating alterations that might prevail in disease.

4. This oblique view has proved of value in over forty-five cases in which the roentgen changes were correlated with clinical observations where routine views had failed.

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IMPROVED METHOD FOR SPLENOPORTOGRAPHY USING BIPLANE SERIALIZED EXPOSURES

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TO ABEATICI and Campi[1,2] belong the credit for the first visualization of the venous portal system after direct injection of a radiopaque solution into the spleen. Their investigations were made on dogs in which the injections were done by inserting a needle through the intact skin into the spleen, and also by exposing the spleen through an abdominal incision and then inserting a needle.

They injected 8 to 10 cc. of joduron 70 per cent (iodopyracet), which flows through the sinusoids of the splenic pulp into the splenic vein and thence into the portal vein. In the liver this vein ramifies like an artery and ends in capillary-like vessels, the liver sinusoids, from which the blood is collected by the hepatic veins and is carried by them into the inferior vena cava.

The most favorable moment for the exposure of the films to demonstrate the intrahepatic portal vessels, according to Abeatici and Campi, is about four seconds after the injection. The contrast medium disappears from the portal venous system in about five to six seconds at which time the liver assumes a uniform greater opacity. A small quantity of the medium usually lingers in the spleen, probably due to effusion into the splenic pulp outside of the sinusoidal system. This small amount of material is gradually absorbed. The amount that remains in the spleen is proportional to the quantity injected.

The purpose of this discussion is to briefly call attention to this technique and to summarize the literature on this method, as well as to describe our technique of employing continuous biplane high speed serialized roentgenography to achieve a more comprehensive visualization of the portal venous system. The biplane roll film cassettes [13,14] make it possible to visualize the tributary system of the portal vein, the portal vein itself, and its many intrahepatic branches in greater detail. Such a series of films can be described as splenohepatic angiography and provide an additional means of making experimental studies on this organ as well as for possible clinical examinations.

The technique we have used in our experiments on dogs is essentially that suggested by Abeatici and Campi. After the dog is anesthetized with nembutal (1 cc. per 5 lb.), it is immobilized in the supine position on an animal board and placed on the roentgenographic table. The animal is carefully positioned so that the entire portal circulation can be roentgenographed on the 9½ by 9½ inch frames of the roll film cassettes. The position and exposure factors are tested by making one exposure in each cassette and developing them before inserting the needle. After viewing these preliminary films, it is then easy to make the necessary adjustments. A 16–18 gauge needle, similar to, but slightly larger than a lumbar puncture needle, is inserted through the upper and left lateral abdominal surface directly into the spleen. It is sometimes difficult to strike the spleen in a dog, as it is a long, sausage-like organ and varies somewhat in its location. In certain instances we found it more efficient to open the abdomen and bring the spleen into view before inserting the needle. In either procedure, the 20 cc. syringe had been previously filled with 10–20 cc. of urokon sodium 70.*

As soon as the needle is accurately placed within the spleen, the operator gives the signal to the roentgen technician and

* Urokon sodium (sodium aceterizoate, 3-acetylamino-2, 4, 6-triiodobenzoic acid), Mallinckrodt Chemical Company, St. Louis, Mo.

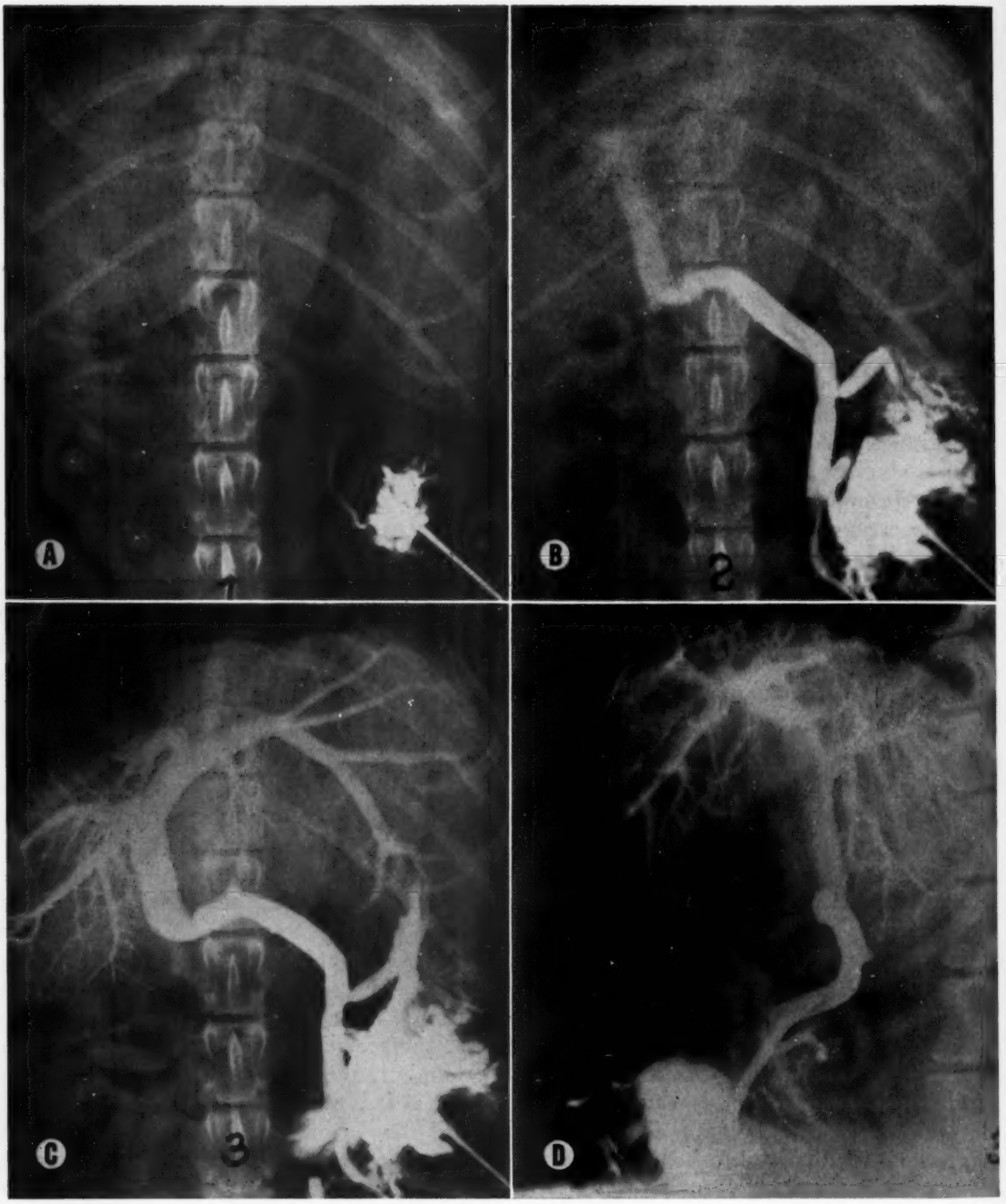

FIG. 1. Splenoportograms using biplane serialized exposures.

(*A*) An 18 gauge needle inserted into the spleen shows the beginning of the injection of 12 cc. of uroken (70 per cent) with a small amount entering the splenic vein.

(*B*) The injection is now over two-thirds completed and the contrast material has filled the sinusoids of the splenic pulp and is flowing out of this organ through three branches of the splenic vein into the portal vein.

(*C*) At this stage the injection has been completed for two seconds and the contrast material continues to flow through the portal vein into the liver where it ramifies like an artery and ends in capillary-like vessels, the liver sinusoids. The material is then collected by the hepatic veins and carried by them into the inferior vena cava. Of these structures, only the inferior vena cava is usually demonstrable.

(*D*) Lateral splenoportogram made simultaneously with the anteroposterior view shown in (*C*). In an organ the size of the liver and with as rich and complex circulation, lateral views are essential for their comprehensive visualization.

two exposures are made by the biplane roll film cassettes before the injection is done. The contrast medium is then quickly injected within two to three seconds with the roll film cassettes in continuous operation making exposures at the rate of two per second for a period of six to ten seconds. By this means both the anteroposterior and lateral views of the portal system are recorded simultaneously. We believe that the right angle views offer a better possibility for a correct interpretation of abnormalities involving the portal system. All the phases, including the filling and emptying of the portal system, can be fully recorded and usually within six seconds from the beginning of the injection (Fig. 1, *A*, *B*, *C* and *D*).

The question naturally arises as to the hazard of the examination and what damage, if any, is inflicted on the spleen either immediately or at a later date. In dogs histologic examinations[2] of the spleen ten to thirty days after the injection did not reveal any significant abnormality other than moderate dilatation of the sinusoids and minor subcapsular hemorrhages. These changes could nearly always be seen in dogs subjected to two or three injections. The follicles remained normal in number and size and the reticuloendothelial system did not show any reaction to the mechanical and chemical stimuli.

Information concerning possible histopathologic changes after similar injections into humans is scarce. Boulvin and his coworkers[4] did not mention this problem, while Dreyer and Budtz-Olsen[6] simply reported that their patients experienced no untoward reactions. Sotgiu and his associates[15] were the only ones to report a case in which the spleen had been injected twice at an interval of twelve days. Twenty-four hours after the second injection a splenectomy was performed and the organ studied grossly. They were able to demonstrate the puncture tract, but without evidence of a subcapsular hemorrhage, or other damage to the spleen. Only slight hemorrhage could be detected microscopically at the injection site.

With further refinement, this method may have applications in the examination of patients with primary diseases that cause a hepatosplenomegaly, and especially in the investigation of hypertensive portal syndromes due completely or partially to obstructive phenomena. These may be extrinsic, such as tumors, adenopathies, perivascular sclerosing processes, or they may be intrinsic, such as venous thrombosis or occlusion. Boulvin *et al.*[4] were the first to do a splenic puncture in a patient with injection of contrast material for the visualization of the portal circulation. They demonstrated an obstruction of the splenic vein, probably due to extrinsic obstruction. The patient was not subjected to operation and no confirmation of the diagnosis was obtained. Dryer and Budtz-Olsen[6] made similar injections in 11 patients, and in 1 case demonstrated an obstruction of the splenic vein which at operation was found to be due to a thrombosis. A splenectomy was performed with relief to the patient. Sotgiu, Cacciari and Frassineti[15] performed this technique in 3 patients with splenomegaly: 1 due to a myeloid leukemia, 1 with Banti's syndrome, and 1 with a hypertrophic cirrhosis of the liver and an enlarged spleen. The portal circulation in these 3 cases showed little variation from the normal. They also performed this procedure in 1 patient in whom they were able to make a diagnosis of thrombophlebitis of the splenic vein and confirmed it by operation.

Moore and Bridenbaugh[10,11] visualized the intrahepatic portal system by inserting a polyethylene catheter into the right gastro-epiploic vein and advancing it into the portal vein where it was tied in place. This technique was performed as an incidental procedure on patients undergoing a laparotomy. With the catheter in place, 20 cc. of 35 per cent diodrast was injected within two to three seconds, followed immediately by a roentgenographic exposure. They obtained satisfactory roentgenograms of the portal vein and radicles in most cases, although the portal radicles in the left lobe of the liver were not filled con-

sistently. No serious reactions or complications developed following this procedure.

Child and his associates[5] used a similar technique in which a needle was inserted directly into the superior mesenteric vein. An injection of 35 per cent diodrast was made quickly and films were taken immediately thereafter. The procedure was performed during an abdominal laparotomy on 21 patients. They concluded that the procedure did not add significantly to the overall surgical risk and that it may be of value in the study of the dynamics of the portal circulation.

From these reports it appears that the actual injection of the contrast medium into the spleen does not in itself cause any damage to the cellular structure. The chief objection to performing this examination on patients is the possibility of incurring mechanical trauma with the insertion of the needle. While no deaths have as yet been reported in the few patients on whom this technique was performed, it cannot be done without some hazard. Experience in taking a liver biopsy with a Silverman needle indicates a mortality rate of about 1 per cent.[7] Similar needle biopsies of the spleen are done in this country, but not with as great frequency.[3] The hazard for both examinations is probably about the same, yet it could be less for the spleen. The spleen is a more highly vascularized organ than the liver, and hemorrhage within its substance or that due to a rent in the capsule with bleeding into the abdominal cavity need not be fatal. If such an accident occurs, a splenectomy can be done.

Thus, splenohepatoangiography should be reserved for the patient in whom the information to be gained will justify the hazard incurred by the examination. It should be pointed out that splenic punctures are performed much more frequently in the European countries[8,9,12] and with much less hesitation than they are undertaken here, a fact which suggests that it may not be as serious a procedure as we have come to believe. Undoubtedly the safety with which splenic punctures are carried out is largely commensurate with the experience and skill of the operator.

CONCLUSIONS

(1) The development and technique of splenoportography have been described.

(2) The technique has been improved by the use of simultaneous biplane high speed, continuous automatic serialization of the roentgenographic exposures with roll film cassettes. This method provides a more comprehensive and detailed visualization of the splenoportal system, including the splenic vein, the portal trunk and the many intrahepatic branches.

(3) Splenoportography is a new technique for the exploration and investigation of these structures and with further refinement can probably be adapted for satisfactory clinical examinations.

(4) The success and safety of the examination depends largely on the skill with which the splenic puncture can be performed.

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EXPERIMENTAL METHOD FOR VISUALIZATION OF THE HEPATIC VEIN—VENOUS HEPATOGRAPHY

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A RENEWED interest has developed in methods for the roentgenographic visualization of the liver by means of radiopaque substances. In general, the problem has been approached by: (1) the intravenous injection of radiopaque chemicals in emulsions, in suspensions or in oils which are removed from the blood stream by the liver cells, and (2) the injection of opaque chemicals into the vascular tree of the liver.

PROCEDURES FOR VISUALIZATION OF THE LIVER PARENCHYMA BY RADIOPAQUE SUSPENSIONS, EMULSIONS AND OILS

One of the earliest clinical methods for radiopacification of the liver has been hepatography with colloidal thorium dioxide. Initially this procedure appeared promising, but has since been largely discarded because of the injurious effects induced by the radioactivity of the particles of thorium dioxide and by their blockage of the reticulo-endothelial system.[6,9] Furthermore, the opacification did not supply a picture of the architectural details of the liver.

In 1939 Degkwitz[2] and Beckermann[1] used a tri-iodide ethyl ester of stearic acid in colloidal suspensions (jodsol) to obtain prompt radiopacification of the liver within about fifteen to thirty minutes after the injection. Unfortunately, they had difficulty in stabilizing the colloid. Since it was a nonstable product and gave rise to toxic reactions, it was discarded. However, in the last year the product has been restudied and the suspension medium altered to render it stable. It is now marketed under the name of "Angiopac."* An intravenous injection of 25 cc. of angiopac in an average size adult will produce a definite, but only a moderate degree of opacification of the liver within fifteen to thirty minutes after the injection. Sufficient time has not yet elapsed to provide enough experience in the use of angiopac to recommend it for clinical examinations. It, like thorium dioxide sol, produces only a homogeneous increase in the density in the liver shadow without providing details in structure.

Olsson and Ekman (4) in 1949 reported the possibility of obtaining hepatograms after the oral ingestion of brominated fatty acids. Such substances have produced opacification of the liver and spleen in rats, but have not been used in man, largely because of the huge quantity of bromine that would be necessary.

In 1942 Newell[3]† suggested the use of suitably emulsified radiopaque oils for hepatography. This idea was later discussed by George and co-workers,[3] but it remained for Thomas and associates[10] to make the first attempt in this direction.

They found serious disadvantages in the oils (iodochloral, lipiodol, and others) due to fluctuations in the particle size of the oil droplets which lead to acute embolic phenomena. By employing the newer types of wetting agents and protective colloids, they were able to obtain oil droplets of more homogeneous size and emulsions of greater stability, but never succeeded in developing an agent for clinical use.

VISUALIZATION OF THE LIVER BY OPACIFICATION OF THE VASCULAR TREE

Radiopacification of the vascular system of the liver provides a more detailed demonstration of the anatomic and pathologic changes caused by displacement, distortion,

* Aqueous emulsion of ethyl-di-iodostearate, Union Chimique Belge, Division Pharmaceutique, 68 Rue Berkendael, Brussels, Belgium.

† In discussion.

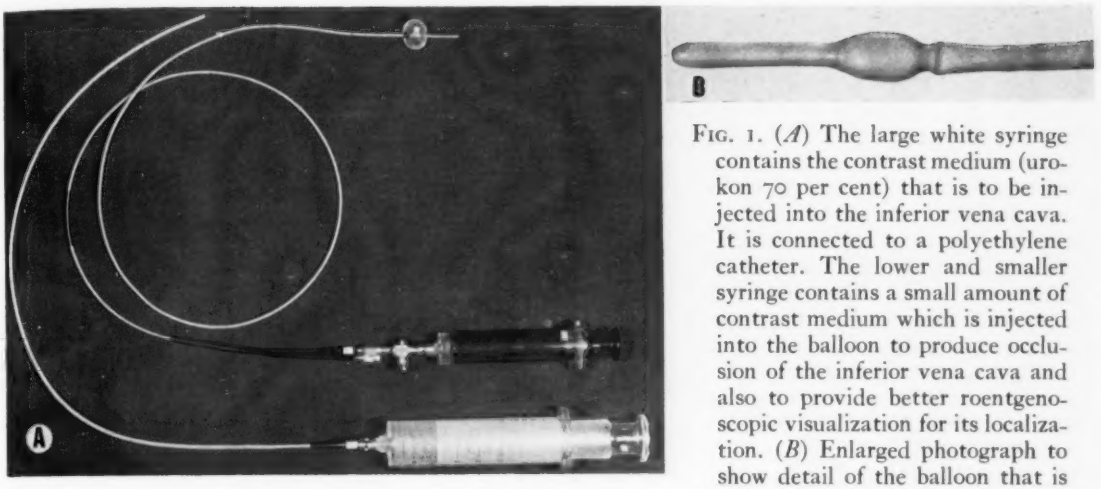

Fig. 1. (*A*) The large white syringe contains the contrast medium (urokon 70 per cent) that is to be injected into the inferior vena cava. It is connected to a polyethylene catheter. The lower and smaller syringe contains a small amount of contrast medium which is injected into the balloon to produce occlusion of the inferior vena cava and also to provide better roentgenoscopic visualization for its localization. (*B*) Enlarged photograph to show detail of the balloon that is fitted to the end of the lateral opening in one end of the lumen in the Cournand catheter.

complete or partial occlusion of the vessels which may be the clue to the underlying pathologic disorder. Three methods are available for the visualization of the vascular system of the liver: (1) injection of the hepatic artery; (2) injection of the portal vein, and (3) retrograde injection of the hepatic vein.

(1) Angiograms of the hepatic artery are occasionally obtained during abdominal aortography. Usually the hepatic artery contains an inadequate amount of contrast medium and the arterial tree is seldom visualized with any degree of completeness. This method cannot be counted on to provide a constant or practical means of visualizing these vessels. However, more complete filling of the hepatic artery is obtained when the injection is made close to the celiac axis.

(2) The portal system can be visualized by direct injection of a contrast medium into one of the tributary veins of the portal system, either during an abdominal operation or by the direct injection of the contrast medium into the spleen by a splenic puncture. In the latter the radiopaque solution flows from the sinusoidal system of the spleen into the splenic vein, enters the portal trunk and flows through the intrahepatic branches of this vessel. The details of the technique of this method were discussed in the preceding article[11] and although it has been used for clinical purposes the method has not yet been perfected.

(3) The retrograde visualization of the venous outflow system of the liver is occasionally obtained during angiocardiographic examinations in children, particularly in those with pulmonary stenosis. Rappaport[5] developed an experimental method in the dog whereby he inserted a special double transflex French No. 10 catheter through the saphenous vein into the inferior vena cava and thence into one of the hepatic veins under roentgenoscopic control. When in proper position the metal guide was withdrawn and 20 cc. of diodrast 70 per cent was injected during which, and shortly after, serial roentgenograms were made to follow the progress of the contrast medium.

This method gives a means for visualization of the venous system within a selective segment of the liver. The catheter used by Rappaport has a small rubber balloon near the tip which can be inflated by injecting 35 per cent diodrast through the smaller tubing to wedge it into the catheterized vein to prevent backflow. After the injection the diodrast spreads through communicating channels with other liver veins to produce a very dense and homogeneous shadow of the injected segment. Rappaport

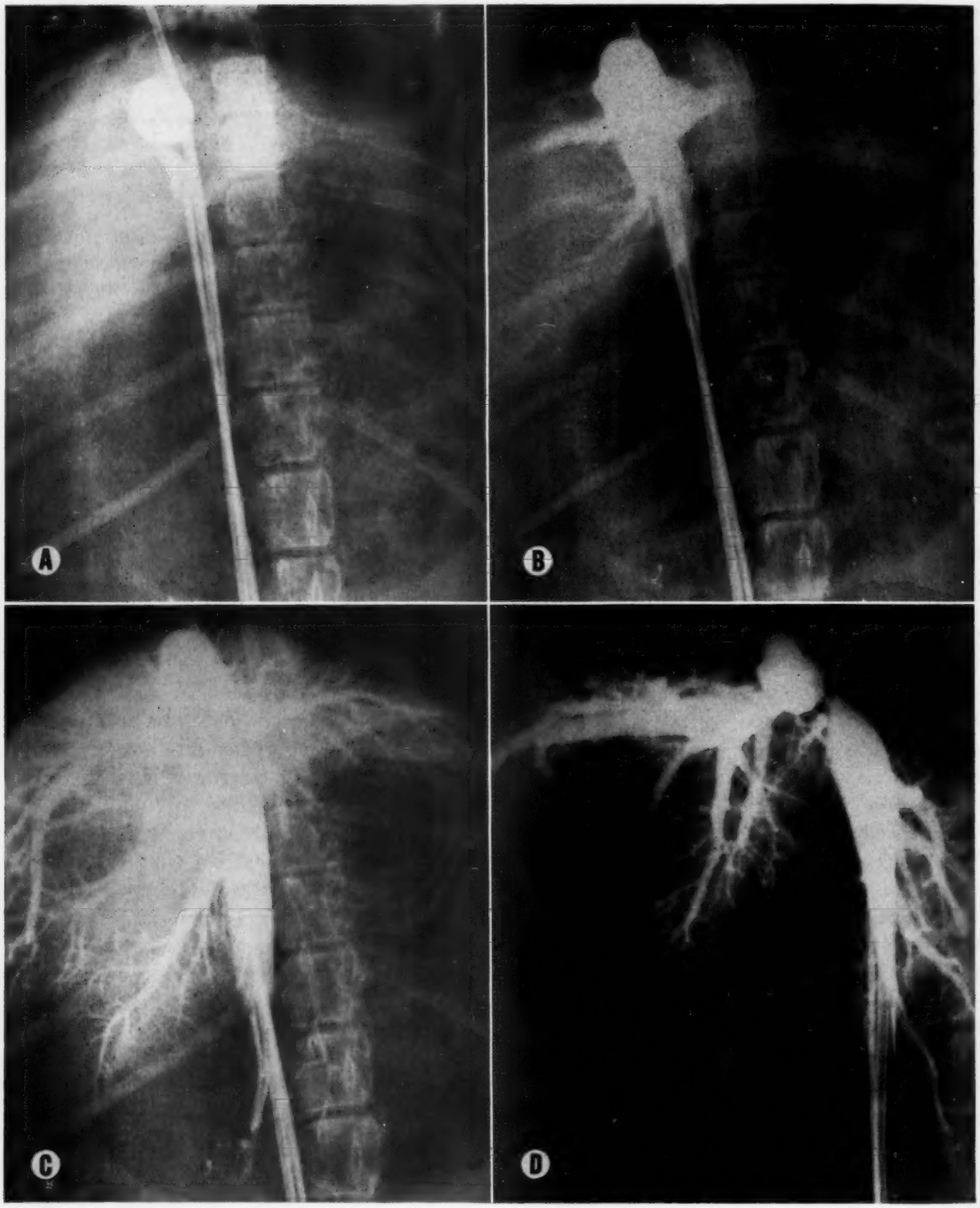

Fig. 2. Retrograde hepatic venograms.

(*A*) A double lumen Cournand catheter equipped with a small balloon is inserted into the femoral vein and fed into the inferior vena cava until the tip is 2 to 3 cm. above the diaphragm. The balloon has been inflated with a few cubic centimeters of urokon to make it easily visualized and to occlude the inferior vena cava. The second catheter of polyethylene has been inserted in the same femoral vein until the tip lies a few centimeters below the balloon.

(*B*) The first of 20 cc. of urokon (70 per cent) has just been injected into the inferior vena cava (1.5 seconds after beginning of injection).

maintains that the injected diodrast circulates through the whole liver and reaches the inferior vena cava through different hepatic veins. From our experience we are not certain that this occurs, for we have not been able to demonstrate it by our method.

One of the authors (G.T.)[12,13] in 1952 was the first to undertake retrograde hepatic venography in patients with and without liver disorders. His method required the introduction of a Cournand catheter into the right or left basilic vein, advanced through the superior vena cava, past the right atrium, through the inferior vena cava and into the main right or left hepatic vein. The passage of the catheter is followed by roentgenoscopic observation. When the tip of the catheter is properly located, 20 cc. of urokon sodium (70 per cent) is quickly injected within two to three seconds. Serialized roentgenographic exposures are started just prior to the injection and continued at one-half second intervals for three to four seconds. This procedure is well tolerated and no accidents or untoward reactions, either immediate or late, have been observed. Both this method and that proposed by Rappaport provide visualization of only about one-half of the hepatic veins and consequently do not give a complete picture of the venous system.

In order to obtain complete filling of all the hepatic veins, we have evolved another method in which the inferior vena cava is temporarily occluded by a small rubber balloon a few centimeters proximal (superior) to the opening of the hepatic veins and before the vena cava enters the right antrium. In this way, contrast medium injected below the balloon would be free to enter all the hepatic veins that open into the inferior vena cava.

The technique requires a double lumen Cournand catheter equipped with a small balloon which is fitted around the lateral opening in the end of one of the lumens (Fig. 1*A* and *B*). With the dog anesthetized the femoral vein is exposed and the catheter with the balloon inserted and fed into the inferior vena cava until the tip is 2–3 cm. above the diaphragm. The placing of the tip of the catheter is determined by roentgenoscopic examination. When it is in the proper location, the balloon is inflated with a few cubic centimeters of contrast medium to make it more easily visualized (Fig. 2*A*). At this time it is also possible to determine the quantity of contrast medium which must be injected into the balloon to occlude the inferior vena cava.

With the balloon correctly located, the catheter is fastened in place with a suture near the site of insertion to immobilize it. A second catheter of polyethylene is introduced in the same femoral vein and directed roentgenoscopically until the tip lies 3–4 cm. below the level of the balloon. Two to 3 cc. of contrast medium should be injected into the catheter to obtain better visualization of it (Fig. 2*A*). A stop-cock attached to the proximal end of the catheter will prevent the contrast medium from draining out. The proximal end of the second catheter is also secured near the site of introduction to hold it in place.

The dog, already tied to the animal board, is placed on the roentgenographic table and properly positioned. Preliminary exposures are made in the anteroposterior and lateral projections as a check on the positions of the dog and the catheters, as well as on the exposure factors prior to injecting the contrast medium.

When the preliminary films are checked and adjustments completed, the operator fills the balloon with opaque material to occlude the inferior vena cava. The automatic biplane roentgenographic unit equipped with roll film cassettes[7,8] is started and, after one exposure, 20 cc. of urokon

(*C*) Complete filling of the hepatic veins including the very small radicles and tributaries (4 seconds after beginning of injection).

(*D*) Lateral retrograde hepatic venogram made simultaneously with the anteroposterior view shown in (*C*) which demonstrates the necessity of right angle examinations for a comprehensive demonstration of the venous system.

70 per cent is rapidly, but not violently, injected through the polyethylene catheter within two to three seconds (Fig. 2*B*). The opaque chemical flows in a retrograde manner to fill the hepatic veins. This provides excellent visualization of the efferent venous system of the liver (Fig. 2*C* and *D*). Roentgenographic exposures are continued for four to five seconds at the end of which the balloon is deflated to allow the retrogradely filled hepatic vein to empty into the vena cava.

We have not applied this method in the clinical examination of patients, although we do not feel that there are any serious technical contraindications. The principal hazard is the same as that which applies to all angiographic examinations in which an opaque chemical is injected into the vascular system. In this procedure, the contrast medium remains within the venous system and does not come directly in contact with the liver cells.

CONCLUSIONS

(1) We believe that our technique for obtaining retrograde hepatograms of the venous system by temporarily occluding the inferior vena cava can be developed into a practical diagnostic procedure of clinical value.

(2) On theoretical grounds, it is reasonable to presume that pathologic processes taking place in the liver which alter the vascular bed within it by displacement, distortion, partial or complete occlusion can be detected and studied to render a more complete and accurate interpretation of disorders within the liver.

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ROENTGEN THERAPY OF MALIGNANT LYMPHOMA WITH SPECIAL REFERENCE TO SEGMENTAL RADIATION THERAPY*

RESULTS 1935–1945

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THIS report represents the experience of the senior author with a consecutive series of all histopathologically proved cases of malignant lymphoma admitted to the Carpenter Memorial Clinic, a tumor clinic in a general hospital treating both service and private patients, from 1935 to 1945 inclusive.

The consideration together of several variants of malignant lymphoma is justified by their similar clinical course and the identical therapy required. Custer and Bernhard[7] report the transition from one type of lymphoma to another in a large series of cases and conclude that they all belong to one disease entity. In a recent case not in this series, follicular lymphoblastoma and Hodgkin's disease were found in different parts of the same lymph node. The frequent transition of follicular lymphoblastoma to lymphosarcoma has been reported. Pathologists frequently differ, particularly when the diagnosis lies between Hodgkin's disease and reticulum cell carcinoma. No attempt has been made to subdivide the Hodgkin's disease cases by Jackson and Parker's method since the clinical course has not been found to correlate too well with these subdivisions. Since therapy yields better results in some forms of lymphoma than others, we have tabulated our cases according to histopathological diagnosis (Tables I and II).

Fifty-two cases of malignant lymphoma with adequate biopsy proof were found. Four patients had follicular lymphoblastoma (2 later underwent transition to lymphosarcoma) of whom 3 survive from six to thirteen years (Table III). Nineteen patients had lymphosarcoma with 7 (37 per cent) five year survivals, of whom 6 are still alive. One died from recurrence in the sixth year (Table IV). Nineteen had Hodgkin's disease with 9 (47 per cent) five year survivals. Five are living now (1 with recurrence in the tenth year). Four died

TABLE I

MALIGNANT LYMPHOMA (1935–1945)
52 cases

	No. cases	Died		Alive 5–15 years		
		Before 5 yr.	After 5 yr.	With recurrence	Apparently well	
Follicular lymphomastoma	4	1	0	1	2	
Lymphosarcoma	19	12*	1	0	6	
Hodgkin's disease	19	11†	3	1	4	
Reticulum cell sarcoma	5	4	0	0	1	
Unclassifiable	5	5				
TOTAL	52	33	4	2	13	(25%)

* Died intercurrent disease, 2.
† Died intercurrent disease, 1, lost 1.

* Presented at the Thirty-third Annual Meeting, American Radium Society, Atlantic City, N. J., June 7–9, 1951.

TABLE II

MALIGNANT LYMPHOMA (1935–1940)
19 cases

	No.	Well 10 Years	
Follicular lymphoblastoma	3	2	
Lymphosarcoma	10	2	
Hodgkin's disease	8	1	
Reticulum cell sarcoma	2	1	
Unclassified	2	0	
TOTAL	25	6	(24%)

from recurrence in the sixth to ninth year after admission (Table v). Five had reticulum cell sarcoma, of whom 1 survives for ten years. Five cases could not be classified since several competent pathologists did not agree on the type of lymphoma present, but the clinical course was that of malignant lymphoma, and none survive (Table VII).

All survival figures are calculated from the date of the first visit and not from the onset of symptoms. Since some authors report survival from onset of symptoms, we have included in our tables the duration prior to admission, from which survival after onset can be computed. In Caulk's[4] recent report the five year survival based on onset of symptoms was 50 per cent, but when calculated from the time of therapy it dropped to 15 per cent.

There are relatively few reports concerning the relative incidence of the histopathologic variants of malignant lymphoma, but the distribution of this series seems about average. The distribution is almost in the same ratio as Gall and Mallory's[9] series of 618 cases. However, Jackson and Parker[11] report over half of their malignant lymphomas to be Hodgkin's disease, as do Merner and Stenstrom.[13] Inferential estimates from other sources[5,14] where both Hodgkin's disease and lymphosarcoma are reported separately indicates that Hodgkin's disease accounts for about half of the malignant lymphomas.

Nineteen of our 52 patients survived more than five years after admission, an absolute five year survival rate of 36.5 per cent. However, 6 patients had known recurrence at the end of the fifth year, of whom 4 have died and 2 continue to require therapy, leaving 13 patients (25 per cent) apparently well six to fifteen years after treatment (Table I). Six of 25 cases treated from 1935 to 1940 were alive and without recurrence at the end of 1950, a ten year absolute survival rate of 24 per cent (Table II).

TABLE III

FOLLICULAR LYMPHOBLASTOMA

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
E.M. No. 52	35	?	Generalized	(1) Previously treated (2) Very low gen. Rx (100–500 r)	18 mo. after admission	Rx inadequate
E.F.* No. 876	28	3 yr.	Retroperitoneal mesenteric, iliac and inguinal. Bulky	(1) Low segm. Rx 1937–42 (200–500 r) (2) Light sympt. 1946–1947 (ad 1,000 r)	13 yr. No recurrence past 3 yr.	Early Rx, inadequate. Later sympt. therapy more adequate. (1946–? transition to LSA)
G.G.B.* No. 1299	28	2 mo.	(1) Retroperit., 1938 (2) R. axilla, 1940	(1) Heavy (2,700 r A and P) to abdomen, 1938 (2) Light (400 r) segmental, 1940	12 yr. No recurrence past 10 yr.	Diagnosis not made until 1940. At first called "paraganglioma"
C.P.† No. 3841	36	9 mo.	(1) Generalized nodes (2) Visceral	(1) Low segmental (ad 500 r) (2) Light symptomatic (ad 1,000–1,200 r)	6 yr.; alive with visceral disease (liver and spleen)	Primary treatment probably inadequate. Transition to LSA

* Well more than five years.
† Alive with recurrence.

TABLE IV

LYMPHOSARCOMA

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
N.T. No. 81	54	0 mo.	Generalized, bulky	Low to light segmental	4 mo.	Advanced disease. Very inadequate therapy
M.B. No. 134	34	42 mo.	Generalized, recurrent visceral	Prior therapy elsewhere. Symptomatic	3 mo.	Too advanced for more than palliation
E.A. No. 709	80	36 mo.	(1) Larynx and neck (2) Generalized	(1) Light to neck (1,000 r) (2) Low segmental (100–400 r)	4 yr.	(a) Initial Rx local (b) Segmental Rx inadequate
R.S. No. 1467	55	1 mo.	Generalized, bulky. Probably visceral	(1) Light segmental (500–1,000 r) (2) Symptomatic	11 mo.	Initial segmental treatment inadequate
E.D.* No. 1657	63	0	L. neck, axilla and groin	(1) Light segmental (500–1,000 r) (2) Moderate therapy 4 yr. later to previously untreated areas	12 yr. (7 yr. since last recurrence)	Recurrence only in untreated segments (R. axilla, 1943)
E.C.† No. 1692	50	1 mo.	(1) R and L neck (2) Generalized	(1) Moderate to neck, light to axilla and mediastinum (2) Moderate to abdomen and groins 1 yr. later (3) Symptomatic	62 mo.	Recurrence in lightly treated (100 r) axilla. Segmental subdiaphragmatic. Rx too late (1 yr. later)
L.D. No. 1794	72	12 mo.	Groins, iliac, aortic ?mesenteric. Bulky	(1) Light partial (subdiaphragmatic) segmental (2) Later symptomatic	41 mo.	Essentially symptomatic therapy (age and radiation sickness)
L.L.* No. 2068	65	12 mo.	Right neck only	Right neck only, heavy (2,200 r)	11 yr. plus	Therapy to obvious nodes only
H.M. No. 2179	55	10 mo.	Generalized, bulky. Inf. vena caval block	(1) Previous inadequate Rx (2) Symptomatic Rx only	5 mo.	Too advanced for any but palliative Rx
L.W. No. 2527	75	4 mo.	Generalized, bulky	Low to light segmental	11 mo.	Sudden death while symptom-free
H.S.* No. 3043	65	6 mo.	(1) R. and L. axilla (2) 6 hr. later generalized, bulky with pemphigus	(1) None. Refused by patient for 6 yr. (2) Symptomatic	8 yr. since diagnosis. 9 yr. since onset. 2 since Rx	Excellent regression of bulky nodes, including some not treated
E.M. No. 3098	22	3 mo.	(1) Vulva and breast (2) Generalized	(1) Symptomatic (2) Segmental (3) Symptomatic	4 yr. after admission. 3½ after treatment	Diagnosis at first obscure and treatment withheld. Autopsy diagnosis LSA with transition to fibrosarcoma
J.D.‡ No. 3137	67	5 mo.	(1) L. neck and mediast. (2) Axillas, mediast. R. neck, groins	(1) Heavy to L. neck; light to mediastinum (2) Gradual segmental irradiation	Died 3 yr. after admission from intercurrent disease. (postop. shock)	Spotty segmental irradiation. No apparent disease at time of death
G.S. No. 3288	44	4 mo.	(1) R and L neck, mesentery, rt. femoral (2) R. neck, bones, retroperitoneal	(1) Partial segmental (2) Rx to nodes in previously untreated areas (3) Symptomatic	4½ yr.	Had moderate Rx to limited areas. Recurrences chiefly in untreated zones
G.K.* No. 3551	39	7 mo.	L. groin and femoral. R. and L. iliac. Mesenteric	(Light segmental (1,000 r) (1) Subdiaphragmatic (2) Supradiaphragmatic (interval between series 1–2 mo.)	6½ yr.	Probably adequate Rx. (1,000 r per field)

TABLE IV—*Continued*

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
H.M.‡ No. 3581	71	3 mo.	Generalized	Partial segmental (light)	11 mo.	Accidental death
J.B. No. 3769	63	36 mo.	Generalized and visceral	Symptomatic	2 mo.	Too advanced. Pulm. tuberculosis with neg. tbc. test
M.H.* No. 4099	39	12 mo.	Right neck	Moderate segmental	5¼ yr.	Apparently cured
T.P.* No. 4242	56	3 mo.	Left axilla (1945). Left femoral (1950)	(1) Mod. segmental except groins (1,200 r). (2) Lower quad., inguinal and femoral	5 yr.	Only recurrence in untreated groin 4¾ yr. after admission

* Well more than 5 years.
† Survived with disease more than 5 years.
‡ Died from intercurrent disease.

TABLE V

HODGKIN'S DISEASE

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
E.H. No. 262	53	14 mo.	Generalized nodes and visceral (recurrent)	(1) Previous therapy elsewhere (2) Symptomatic therapy only	17 mo.	Recurrent after prior treatment. No serious attempt at other than palliative treatment
T.C.* No. 333	18	12 mo.	Left neck, mediatinum, R. and L. axilla	(1) Light segmental to upper trunk (1,000 r). Low to lower trunk (400 r)	15 yr.	Probably adequate therapy above diaphragm, of doubtful value below
M.S.† No. 343	29	13 mo.	R. and L. cervical, L. axillary, R. groin, retroperitoneal, general pruritus	(1) Low to light segmental (400–700 r) (2) Symptomatic for recurrences	5 yr.	Tolerated therapy poorly. Dosage inadequate
S.S.† No. 556	12	36–48	R. cervical, mediastinal and R. axillary. Later visceral and generalized nodes	(1) Light therapy to proven disease. Very low (100–200 r) to other segments (1936) (2) Symptomatic (1937–1941)	5 yr.	Initial therapy very inadequate segmental irradiation
G.G. No. 1168	23	72 mo.	R. and L. neck, R. axilla, mediastinum and L. lung, spleen	(1) Therapy elsewhere for 5 yr. (2) Symptomatic treatment only	16 mo.	Advanced, recurrent disease. Marked subcutaneous and pulmonary fibrosis. Respiratory death
H.N.‡ No. 1474	34	1 mo.	R. neck (1938); ? spleen (1939); ? lung (1939)	(1) Moderate to R. neck. Light to mediastinum (2) 1 yr. later light to spleen, epigastric and iliofemoral	2 yr.	Died from miliary tuberculosis. At autopsy no Hodgkin's found
M.B. No. 1556	46	16 mo.	Generalized nodes, visceral, pruritus (recurrent)	(1) Prior treatment elsewhere (2) Symptomatic (9 Rx)	4 mo.	Advanced, recurrent disease. Had had extensive prior Rx elsewhere
V.E.† No. 2112	31	4 mo.	(1) L. supraclavicular and L. hilar (1940) (2) R. and L. neck, mediastinum, R. and L. axilla, spleen (1942)	(1) Moderate to 2 small fields (1940) (2) Light to moderate segmental (1942) (3) Symptomatic (1943–1945)	5 yr.	Incorrect diagnosis of tuberculous lymphadenitis (inadequate biopsy) delayed segmental therapy for 2 yr.

TABLE V—*Continued*

Identity	Age	Duration	Extent	Treatment	Survival	Comment
T.N.* No. 2644	16		Bulky L. cervical only	(1) Light segmental above diaphragm (1,000 r) (2) 8 mo. later, moderate segmental below diaphragm and spleen (1,200 r)	9¾ yr.	Adequate therapy but long interval between upper and lower series
J.S.† No. 2741	5	6 mo.	(1) R. and L. neck (1941) (2) Spleen (1946) and generalized	(1) Light segmental (800 r) divided series over 9 mo. omitting spleen	8 yr.	Extreme youth of patient encouraged very cautious gradual therapy
J.H.§ No. 2842	17	100 mo.	1941 generalized, very bulky nodes and spleen. 1946 ? mediastinum. 1951 multiple nodes	(1) Mod. segmental to ant. trunk, did not complete posterior (2) 1946 moderate rt. hilar A. and P	9 yr. 9 yr.	Hodgkin's disease 8 yr. before therapy. Failed to follow prescribed Rx. Living with disease and responding to Rx 9 yr. after initial therapy
H.H. No. 2872	45	4 mo.	(1) Mediastinal, retroperitoneal and visceral, fever and malaise	(1) Moderate mediastinal (2) Mod. segmental, lower trunk	9 mo.	Diagnosis made at autopsy. No superficial nodes. Died from HD infiltrating kidney with uremia and cerebral hemorrhage
F.M. No. 2903	21	12 mo.	R. and L. cervical, spleen	(1) Prior therapy elsewhere (2) Light segmental upper trunk, low to lower trunk	11 mo.	Recurrent after prior inadequate therapy. Refused to complete segmental therapy
M.W.* No. 2970	15	11 mo.	L. cervical only	Moderate (1,800 r) to L. neck and upper trunk (1,200 r). Light to lower trunk (1,000 r) 4 mo. later	9 yr.	Adequate therapy. Small, thin patient
H.T. No. 2997	37	0	(1) R. cervical and mediast. (2) R. and L. cervical, hilar, and pulmonary recurrences	(1) Mod. segmental irrad. (1,200 r) upper trunk, followed by light (1,000 r) to lower trunk (2) Symptomatic	2⅔ yr.	Recurrence in treated areas and adjacent viscera (lungs)
J.S. No. 3241	51	8 mo.	Bulky nodes, R. axilla, R. and L. groins, retroperitoneal pruritus	(1) Moderate to heavy segmental therapy to lower trunk (1,600–1,800 r) (2) Symptomatic	11 mo.	Disease never controlled; resistant or too obese for adequate depth dose. Respiratory death
C.L.‡ No. 3362	34	7 mo.	R. cervical and mediast.	(1) None for 6 mo. (2) Light segmental, upper trunk	?	Refused biopsy for 6 mo. Disappeared from observation 6 mo. after initial treatment
C.S.* No. 4077	35	15 mo.	R. cervical	Moderate to R. neck (1,400 r). Light to all segments (1,000 r)	6½	No treatment for 11 mo. after biopsy
L.R. No. 4213	70	1 mo.	R. cervical, R. and L. inguinal, mediastinal, visceral	Heavy symptomatic	10 mo.	Refused biopsy. Biopsy and treatment 5 mo. after first visit

* Well more than 5 years.
† Survived more than 5 years with disease.
‡ Intercurrent disease or lost.
§ Alive with recurrence.

Once again comparable survival figures for a mixed series are hard to find. However, Gall and Mallory[9] indicate that their total group had a survival rate of 20 per cent (apparently from onset of symptoms and not from treatment). Merner and Stenstrom[13] report a five year survival rate of 11 per cent in untreated cases and of 33 per cent in treated cases, also apparently calculated from the onset of symptoms.

Peters[14] recently reported a series of 113 patients with Hodgkin's disease. Her cases were subdivided according to the stage or extent of disease on admission. Stage I (30

TABLE VI

RETICULUM CELL SARCOMA

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
C.M. No. 1095	59	2 mo.	R. axilla, mesentery, liver, spleen	Symptomatic	1½ mo.	Biopsy: follicular lymphoblastoma. Autopsy: reticulum cell sarcoma
H.R.* No. 2356	51	9 mo.	L. inguinal and iliac	Heavy local (3,000 r) to ilio-inguino-femoral	10 yr.	Initial diagnosis malignant tumor, possibly melanoma, later revised to reticulum cell sarcoma
S.M. No. 3157	11	1 mo.	Generalized	Symptomatic	2 mo.	Acute illness, rapidly fatal
H.Po. No. 4179	28	?12 mo.	Mesentery, diffuse retroperitoneal, colon	Hemicolostomy right. No irradiation	½ mo.	
H.Pa. No. 4296	52	6 mo.	(1) R. orbit (2) R. cervical (3) Brain	Exenteration of orbit. Symptomatic irradiation	22 mo.	

* Well more than 5 years.

per cent of the entire series), with involvement of one lymph node group or organ, yielded 88 per cent five year survival. Stage II, with two or more adjacent lymph node groups involved, yielded 72 per cent and Stage III, with involvement of both upper and lower trunk nodes, yielded 9 per cent. Her absolute five year survival rate was 51 per cent for the entire series. While one reason for her excellent results may be the large proportion of early cases (30 per cent compared to an average of around 5–10 per cent) even those in Stage II did much better than a series of cases of lymphosarcoma limited to the head and neck in which Catlin[3] reported a five year survival of 52 per cent. There is no indication that one would expect a lower percentage of survivals in lymphosarcoma than in Hodgkin's disease. Unfortunately, it is not possible to analyze Craver's[5] and Stout's[15] series according to Peters' stages, and thus have a better basis for comparison than over-all results. The impression remains that Peters has better results than any other reported series in spite of criticism[8] that might be raised. The explanation may well be that in most of her cases grossly uninvolved lymph nodes were treated, particularly those adjacent to the site of disease. She demonstrated in her own series a 17 per cent improvement in patients in Stage II and III who had prophylactic irradiation of proximal lymph nodes compared to those who did not.

Since 1935 the senior author had believed that treatment limited to grossly involved lymph nodes is inadequate therapy for malignant lymphoma. Gilbert's[10] report at the Fifth International Congress of Radiology in 1937 supported this impression since Gilbert with segmental radiation therapy achieved a five year survival rate of 34 per cent, distinctly better than the results reported by Craver,[5] Stout,[15] and very much better than several published in the past year.[1,2,4] The present series, if subdivided into stages according to Peters, has had—by the use of the type of therapy which is described below—the following five year results: Stage I 80 per cent, Stage II 55 per cent, and Stage III 17 per cent. Prior to considering further details of the present report, it may be recalled that Medinger and Craver[12] reported better results in Hodgkin's disease with total body irradiation (Heublein technique) than by routine treatment of grossly involved areas of disease only. Their feeling that toxic effects of so much therapy outweighed its advantages may be partly answered by the segmental approach to treatment, in which the more likely sites of disease are irradiated without treating the whole body.

TABLE VII

MALIGNANT LYMPHOMA
Unclassifiable

Identity	Age	Duration	Extent	Radiation Treatment	Survival	Comment
M.K. No. 1560	52	12 mo.	Mesenteric (25×15 cm.). Later cervical, axillary, mediastinal and iliac nodes and pleura	Symptomatic to various masses as they appeared	6 mo.	Radiosensitive but recurred rapidly. Diagnosis by various pathologists, LSA; RCSA
F.K. No. 2533	54	7 mo.	R. cervical and supraclavicular, retroperitoneal. ? thyroid	Symptomatic to various masses as they appeared	19 mo.	Diagnosis by various pathologists: HD, LSA, RSA, Ch. inflam. (6 specimens, first 3 ch. inflammation)
H.N. No. 2730	51		Cervical, supraclavicular, retroperitoneal	Symptomatic	7 mo.	RSA or LSA or anaplastic carcinoma. First positive biopsy 5 mo. after admission
N.G. No. 3238	54	2 mo.	Generalized nodes, spleen, fever, sweats	Light segmental (700 r) anterior fields only	5 mo.	HD? LSA?
A.H. No. 3334	72		R. and L. cervical, thyroid, mediastinal, inguinal	Symptomatic		"Malignant tumor" LSA (Lymphoepithelioma? anaplastic thyroid cancer?) Radiosensitive

TECHNIQUE OF TREATMENT

Roentgen rays, generated at 200 kilovolts (peak), with 0.5 mm. copper filtration, were used at 50 cm. target skin distance for superficial lymph nodes, and at 70 cm. for deep lymph nodes (occasionally at 100 cm. in obese patients). With these factors, the half-value layer is 0.9 mm. of copper. The fields treated varied somewhat in size, ranging from 150 to 250 square centimeters, averaging about 200 sq. cm. each. For fields of this size backscatter is about 40 per cent. Patients were treated five or six days weekly, at the rate of 100 to 200 roentgens through each of two such fields.

The total dose per field varied widely and is discussed below under the four techniques employed. Dosage is expressed as roentgens in air without backscatter.

Segmental therapy is the term used to describe irradiation of all the major lymph node areas: cervical, axillary, mediastinal, mesenteric, retroperitoneal aortic and iliac, inguinal and femoral lymph nodes. In a few cases, the spleen was also included. This plan of therapy was devised in 1935 in the hope of improving the results of therapy, and was used whenever possible. When segmental therapy is used, it is often desirable to treat half the lymph node areas in a single series, and after an interval of one to three months to complete the planned therapy in a second series.

Partial segmental therapy is defined as such treatment to the lymph nodes of half the body, above or below the diaphragm.

Symptomatic therapy is defined as treatment to the lymph nodes producing the most distressing symptoms. Such treatment is used for very ill patients and for those with recurrence after segmental therapy.

Local therapy is defined as treatment to a single group of grossly diseased lymph nodes.

In the treatment of our early cases we were influenced by the then current conviction that low dosage for palliation represented the optimum treatment of lymphoma; gradually we increased dosage in the hope of obtaining cures. For descriptive purposes we define *low* dosage as 100 to 500 r per field, *light* dosage as 600 to 1,000 r, *moderate* dosage as 1,100 to 1,700 r, and *heavy* dosage as 1,800 to 3,000 r per field.

SURVIVAL WITH RESPECT TO TYPE OF THERAPY

No Treatment. One patient, after hemicolectomy for obstructing reticulum cell sarcoma and extensive retroperitoneal and mesenteric infiltration, was considered too

advanced for treatment and died two weeks later.

Heavy Local Therapy (to a single group of nodes). One patient had heavy (2,200 r) therapy to a large cervical field and has had no recurrence in over eleven years. She was considered too feeble for segmental irradiation and would have had symptomatic treatment had the lymphosarcoma spread elsewhere. A second patient had heavy (3,000 r) to the left ilio-inguino-femoral complex of lymph nodes under the mistaken diagnosis of metastatic melanoma. His reticulum cell sarcoma has not recurred after more than ten years.

Symptomatic Therapy (to individual lymph nodes or groups of lymph nodes). Eighteen patients were so treated, 6 of whom had recurrent disease after prior therapy elsewhere. Seventeen patients had generalized disease: 1 had a reticulum cell sarcoma of the orbit with local metastasis recurrent after surgical treatment; 1 patient is living eight years since the diagnosis was made by biopsy, but refused treatment until two years ago; 1 patient was treated under the mistaken diagnosis of tuberculous adenitis for two years and then had symptomatic therapy for Hodgkin's disease, surviving slightly more than five years; the remaining 16 patients died from disease in one to forty-one months after admission.

Partial Segmental Therapy (to the lymph nodes of upper or lower trunk). Eight patients had this primary treatment. One died from miliary tuberculosis without evidence of recurrent Hodgkin's disease (autopsy) two years later. His primary disease was confined to the right cervical nodes. One (Hodgkin's disease) was lost to follow-up after six months, having refused further treatment. One (lymphosarcoma) died from an accident in the first year. Five died from disease, of whom 2 had uncontrollable, radioresistant lymphomas and 3 had recurrence in untreated or lightly treated segments, 1 of whom (lymphosarcoma) survived almost six years.

Segmental Radiation Therapy (to all major lymph node areas). Twenty-three patients had such treatment. Originally we dared give only very low dosage, hence many patients received less than 500 r per field. We now believe that 1,000 to 1,200 r should be given to all fields and from 1,200 to 1,800 r to the grossly diseased lymph nodes. These cases will be considered in two categories:

Light Segmental Therapy (up to 1,000 r per field). Sixteen patients were so treated. Six are still well six to fifteen years after therapy and 3 others survived more than five years. All of these 9 patients had involvement of several groups of lymph nodes. Of the 7 who died within five years, 2 had intercurrent disease and 2 had very inadequate (100 to 500 r) segmental therapy.

Moderate segmental therapy was given to 7 patients. They received 1,000 to 1,200 r to all the major lymph node areas and sometimes up to 1,800 r to the grossly diseased lymph nodes. Five of them are well five to ten years. These 5 had disease apparently confined to one group of lymph nodes, yet 1 case later required therapy to a distant untreated lymph node omitted from the treatment schedule. One other patient has recurrence under treatment in his tenth year. This patient, with generalized bulky Hodgkin's disease, refused to continue a long therapy schedule and had recurrence in an untreated axilla. One patient died in thirty months from recurrence in treated mediastinal lymph nodes.

A daily dose of 200 r through each of two fields of 150 to 200 sq. cm. is usually tolerated by the average patient. Partial segmental therapy (to the lymph node groups above or below the diaphragm) can be continued to 1,200 r or more through each of six such fields. We now try to reach 1,600 or 1,800 r through fields over the deep lymph nodes in the more obese patients. It should be noted that the plan of treatment provides for two opposing fields over the deep lymph nodes (mediastinal, upper axillary, retroperitoneal aortic and iliac), while superficial lymph nodes are treated

through one field only (cervical, lower axillary, inguinofemoral) (Fig. 1). It is generally necessary to discontinue treatment at the end of the first series because of leukopenia and/or radiation sickness. After an interval of one to three months the other half of the body may safely be treated to complete segmental radiation therapy. We consider a white blood cell count of 2,000 or below as an indication to interrupt treatment, but in one case a count of 900 returned to normal without grave complications. Sufficient unirradiated bone marrow is present to prevent agranulocytosis if only half of the body is treated in one series.

We consider it most important to give adequate therapy to the deep lymph nodes, *especially the mesenteric and retroperitoneal aortic and iliac nodes*, which may be involved long before a clinical diagnosis can be made. Whenever the supraclavicular fossa of either side contains lymph nodes involved by lymphoma, we consider this pathognomonic of either mediastinal or retroperitoneal malignant lymphoma, since in our experience such supraclavicular involvement has always been associated with malignant lymphoma of the deep lymph nodes.

We are convinced that treatment to grossly uninvolved lymph nodes is necessary if long term palliation or cure is to be obtained. Eight patients in good general condition, with apparently limited lymph node involvement, were not given adequate segmental irradiation, and subsequently developed recurrence in untreated or lightly treated lymph nodes. Three of these are now well after further therapy, 1 is under treatment for recurrence, and 4 have died. These cases, almost one-sixth of the entire series, are described below.

Case I. G.G.B. (CMC No. 1299) This patient had heavy roentgen therapy (2,700 r×2) to a large retroperitoneal mass under the mistaken diagnosis of paraganglioma. Two years later biopsy from the right axilla showed follicular lymphoblastoma, and comparison with the original biopsy confirmed this diagnosis.

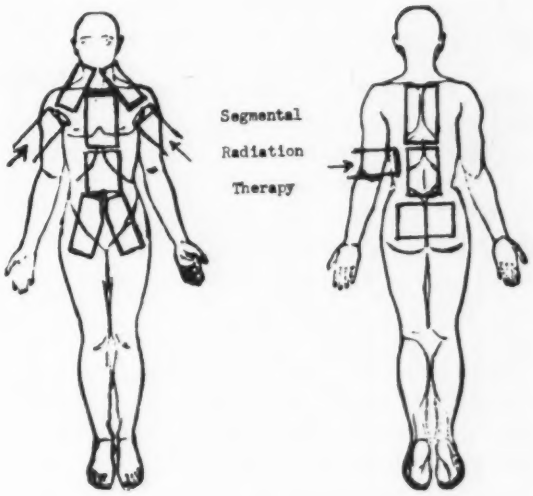

Fig. 1

There were no other palpable lymph nodes. This patient was then given light segmental therapy to the lymph nodes above the diaphragm (omitting the heavily treated abdomen) and has remained free of recurrence for more than ten years.

Case II. E. D. (CMC No. 1657) This patient had lymphosarcoma involving the left cervical, left axillary and left inguinal nodes. He was treated by the segmental technique, including all the deep lymph nodes, but omitting the right neck and right axilla from the treatment schedule. Four years later recurrent lymphosarcoma of the right axilla required treatment. He remains well seven years since his only recurrence.

Case III. E. C. (CMC No. 1692) This patient had *moderate* therapy to the neck and *light* therapy to the axillae and mediastinum for a lymphosarcoma apparently confined to the cervical lymph nodes. Within a year she had recurrence in the lightly treated right axilla and the untreated abdominal lymph nodes. She died from recurrence in her sixth year, in spite of repeated symptomatic therapy.

Case IV. V. E. (CMC No. 2112) This patient had local roentgen therapy to the supraclavicular and left hilar lymph nodes under the mistaken diagnosis of tuberculous adenitis. Two years later partial segmental and symptomatic roentgen therapy controlled her recurrent, generalized Hodgkin's disease for more than three years.

Case v. J. S. (CMC No. 2741) This five year old child with Hodgkin's disease, apparently confined to the right and left cervical lymph nodes, had *light segmental* radiation therapy (up to 800 r) which was gradually completed over a period of nine months. Five years later he had recurrence in the untreated spleen, thereafter in many lymph nodes, and died of recurrent disease eight years after admission.

Case vi. J. H. (CMC No. 2842) This patient had bulky, generalized Hodgkin's disease of eight years' duration. (He had had a neck dissection for localized Hodgkin's disease in 1933.) He received light to moderate segmental radiation therapy, but failed to complete the prescribed series. In his fifth year a hilar recurrence was easily controlled. In his tenth year he is under treatment for recurrence in an untreated axilla.

Case vii. J. D. (CMC No. 3137) After partial segmental radiation therapy (upper trunk) for lymphosarcoma, apparently confined to the cervical nodes, right and left, recurrence in the abdominal nodes required treatment. The patient died from intercurrent disease three years later.

Case viii. N. G. (CMC No. 3238) This patient had partial segmental radiation therapy (upper trunk) for lymphosarcoma grossly involving the parotid and upper cervical lymph nodes. A year later recurrence in the retroperitoneal lymph nodes required treatment. She died four years after admission.

Case ix. T. P. (CMC No. 4242) This patient had lymphosarcoma which appeared to be confined to the left axilla. There were no other grossly enlarged lymph nodes. He had moderate segmental radiation therapy but because of his long torso the treatment did not include the inguinal and femoral lymph nodes. Four years later an untreated femoral lymph node required local therapy. He has had no other recurrence.

SURVIVAL WITH RESPECT TO ANATOMIC EXTENT OF DISEASE

It is generally agreed that the prognosis is better in patients who have lymphoma confined to one lymph node area than in those with generalized malignant lymphoma. Some cases of malignant lymphoma seem to be multicentric from the onset, while others probably originate in one lymph node and may remain localized for months or years. The cases apparently cured by radical lymph node dissection or by local irradiation, such as our cases No. 2068 (lymphosarcoma) and No. 2356 (reticulum cell sarcoma), fall in this category. If we had infallible diagnostic criteria to prove the strict localization of malignant lymphoma, such local therapy would be ideal. Since we can never prove the absence of distant lymph node involvement, we believe even the patient with apparently limited disease should have the benefit of segmental radiation therapy if his condition permits.

Ten of our patients had demonstrable involvement of one lymph node group only. In 6 cases, one cervical group was involved and 5 survive five to ten years, 1 with only local radiation therapy, the others with segmental therapy. The single death was from miliary tuberculosis. One case of ilioinguinal reticulum cell sarcoma survives more than ten years after local therapy. One case of left axillary lymphosarcoma, treated segmentally, had left femoral recurrence four years later in an untreated lymph node. One case of mediastinal Hodgkin's disease died in nine months from retroperitoneal and renal extension, present but unrecognized on admission.

Eleven patients had demonstrable lymph nodes in two anatomic sites, of whom 2 survive, 4 others lived five to eight years, and 5 others failed to survive five years. One survivor had follicular lymphoblastoma twelve years ago and 1 had lymphosarcoma untreated for six of his eight year course since admission: each obviously mild cases of malignant lymphoma.

Two patients with three and four groups of grossly diseased lymph nodes survive fifteen years (Hodgkin's disease) and twelve years (lymphosarcoma) respectively after segmental radiation therapy.

Twenty-nine patients had generalized

malignant lymphoma, many with visceral involvement, fever, anemia, and general malaise. Eight lived less than six months, 6 died in the second six months, 3 in the second year, and 7 more before the fifth year. One case of Hodgkin's disease died after surviving more than five years and 4 patients survive six to thirteen years, 1 of whom has recurrent Hodgkin's disease in his tenth year.

AGE AND SURVIVAL

The only patient in the first decade survived eight years. Of 6 in the second decade, 4 are alive and 1 died after surviving five years. Of 7 cases in the third decade, 2 are well and 1 survived more than five years. Of 10 in the fourth decade, 4 are well and 1 survived more than five years. Of 4 in the fifth decade, none survived. Of 13 in the sixth decade, 2 are well and 1 survived five years. Of 6 in the seventh decade, 3 are well. Of 5 in the eighth decade, none survived five years.

We think the younger patients, in general, have responded better to treatment. Perhaps we tend to give them more persistent therapy because of their greater recuperative powers, possibly the leanness of the younger patient makes for better dosage to the deep lymph nodes. The series is too small to draw valid conclusions.

SUMMARY

A consecutive series of fifty-two histopathologically proved cases of malignant lymphoma is reported. The reasons for considering together the several types of malignant lymphoma are discussed. Absolute survival rates for five years (36.5 per cent) and ten years (24 per cent) are calculated. The technique of segmental radiation therapy and its advantages are described. More aggressive treatment is recommended. A tabulation of all cases is included.

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RADIOACTIVE GOLD-198 FOR GAMMA RADIATION THERAPY*†‡

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PREVIOUS studies at this Medical Center[2,3,17,18,22–26] have demonstrated that the physical properties of the long-lived radioactive isomer of cobalt-60 are superior to those of radium for the preparation of applicators for use in interstitial radiation therapy. For some purposes the properties are sufficiently advantageous over those of radium to permit the fabrication of new types of sources[17,18,23,24] in which radium cannot be used similarly so that new opportunities are presented to explore the application of interstitial radiation sources in ways not previously available.

In the present study, the applicability of the relatively short-lived radioactive isotope of gold, Au^{198}, for the preparation of permanent seed implants as an advantageous substitute for radon, and of removable linear sources for interstitial gamma radiation therapy is considered.

Applications of these gamma-ray sources interstitially in preliminary animal studies demonstrated that they produced regressions in transplanted tumors.[28]

PHYSICAL PROPERTIES OF RADIOACTIVE GOLD-198

Discovery. Gold-198 is an artificial radioisotope of atomic mass 198 and atomic number 79. It was first described in 1934 by Fermi *et al.*[10] who produced it by the interaction of neutrons, generated in a radon-beryllium source, on gold foil, probably according to the reaction,

$$_{79}Au^{197} \xrightarrow{n,\gamma} {}_{79}Au^{198}$$

Neutron Cross-Section. Because of the large capture cross-section for slow neutrons of Au^{197} the only natural isotope of gold, and the availability of gold in high purity,* radioactive gold-198 is generated conveniently in nuclear reactors with very high specific activities† and with little contamination with other radioisotopes. The capture cross-section of gold for thermal neutrons (2,200 m/sec.) is about 95 barns,[30] and Havens *et al.*[13] found a resonance peak of about 3,000 barns at 4.8 electron volts.

Decay Scheme. The decay scheme of Au^{198} according to Mitchell[21] is depicted in Figure 1 where the radioisotope is shown to decay by emission of a beta particle with a maximum energy of 0.97 mev. to an excited state of mercury-198, followed by the emission of a gamma photon with energy of 411 kev. to reach the ground state of mercury-198. In 1950, Hill and Mihelich[15] and Hill[14] reported that 158.5 kev. and 208.5 kev. gamma rays present in gold activated in the nuclear reactor at the Argonne National Laboratory were due to

* The mint gold used in this study was 999.75 purity according to the supplier, The American Platinum Works, Newark, New Jersey.

† The neutron fluxes presently available are sufficiently high to insure that specific activities can be generated readily that are adequate for most purposes in therapy. They range from about 3,000 millicuries/gram[16] in the present nuclear reactor at the Oak Ridge National Laboratory to as high as 180,000 millicuries/gram of gold[8] in the high neutron flux in the Canadian reactor at Chaulk River.

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† Taken largely from theses presented by Lieutenant Benjamin H. Colmery, Jr., USN and by Commander Waldron M. McLellon (CEC) USN to the Graduate School of The Ohio State University in partial fulfillment of the requirements for the degree of Master of Science in the Department of Physics. Commander McLellon and Lieutenant Colmery attended The Ohio State University under the supervision of the United States Naval Postgraduate School, Monterey, California.

‡ Presented at the Thirty-Fourth Annual Meeting, American Radium Society, Chicago, Ill., June 8–10 1952.

** Julius F. Stone Research Associate Professor of Medical Biophysics.

the decay of Au^{199} formed by successive neutron capture by Au^{197}. They reported that Au^{198} has the very high, slow neutron capture cross-section of 16,000 barns so that one might expect to find a measurable quantity of Au^{199} in any sample of gold irradiated in a pile. For gold activated at Argonne[15] the ratio of the numbers of gold-198 atoms to gold-199 atoms was about 95/1, and the ratio of the 0.411 mev. gamma intensity to the 0.159 mev. gamma intensity was about 750/1. Because these ratios are large and the half-life of Au^{199} is only 3.3 days, the radiation from Au^{199} present in gold irradiated in a nuclear reactor is insignificant for the present purposes and it will be given no further consideration here.

Decay Rate. In Figure 2 is shown the decay obtained with an electroscope for a specimen of gold irradiated in the nuclear reactor at the Oak Ridge National Laboratory.[5] It will be seen that the half-life found here is 2.7 days, in good agreement with Saxon and Heller[33] and Silver,[34] who found the half-life to be 2.69 ± 0.01 days. The average life is 3.9 days, or 93.5 hours.

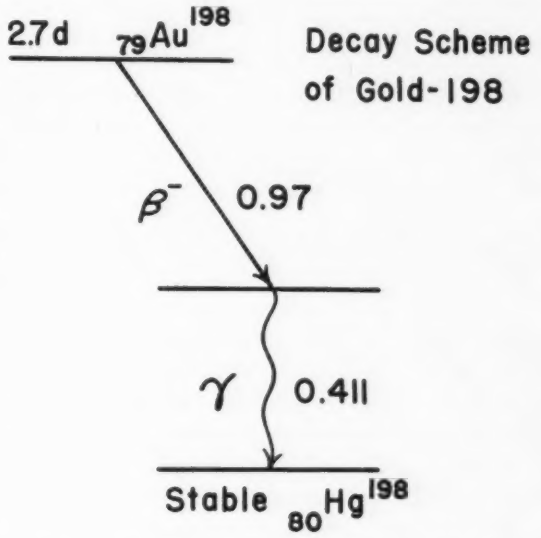

FIG. 1. Decay scheme of radioactive gold-198 (after A. C. G. Mitchell).

Calculated percentages of the initial activity remaining, together with the reciprocals, after intervals of time likely to be of interest in the application of radiogold in therapy are listed in Table 1.

Gamma-Ray Absorption. The experimental mass absorption coefficient in lead

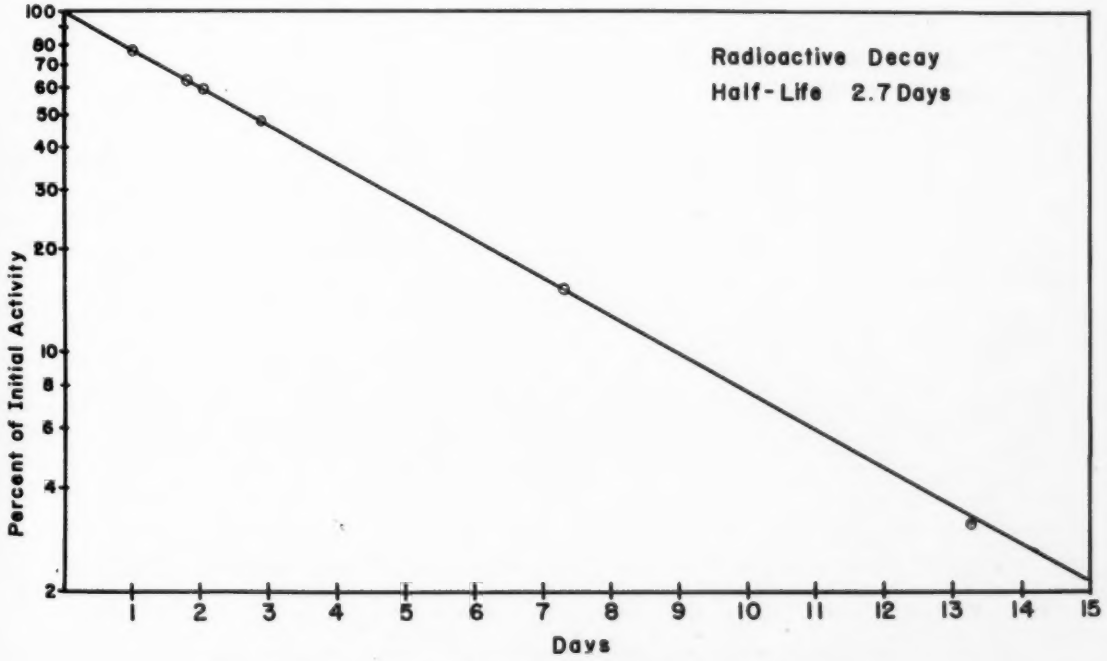

FIG. 2. Decay curve for a radioisotope with half-life of 2.7 days.

TABLE I

PERCENTAGES AND RECIPROCALS OF THE INITIAL ACTIVITY REMAINING AFTER VARIOUS TIMES, CALCULATED FOR A RADIOISOTOPE WITH HALF-LIFE, $t_{\frac{1}{2}} = 2.7$ DAYS $= 64.8$ HOURS

Per cent remaining at time, t(hours) = antilog(2.000000−0.004646t)

t(days) = antilog(2.00000−0.11149t)

Time		Total Hours	Per Cent Remaining	Reciprocal	Time		Total Hours	Per Cent Remaining	Reciprocal	Time		Total Hours	Per Cent Remaining	Reciprocal
Days	Hours				Days	Hours				Days	Hours			
	0	0	100.00	1.000		20	92	37.37	2.676		4	196	12.28	8.141
	1	1	98.94	1.011		22	94	36.58	2.734		6	198	12.03	8.316
	2	2	97.88	1.022	4	0	96	35.81	2.793		8	200	11.77	8.496
	3	3	96.86	1.032		2	98	35.05	2.853		10	202	11.50	8.680
	4	4	95.81	1.044		4	100	34.31	2.915		12	204	11.28	8.867
	5	5	94.79	1.055		6	102	33.58	2.978		14	206	11.04	9.059
	6	6	93.78	1.068		8	104	32.87	3.042		16	208	10.81	9.255
	7	7	92.79	1.078		10	106	32.18	3.108		18	210	10.58	9.455
	8	8	91.80	1.089		12	108	31.50	3.175		20	212	10.35	9.659
	9	9	90.83	1.101		14	110	30.83	3.244		22	214	10.13	9.868
	10	10	89.85	1.113		16	112	30.18	3.314	9	0	216	9.92	10.08
	11	11	88.90	1.125		18	114	29.54	3.386		2	218	9.71	10.30
	12	12	87.95	1.137		20	116	28.91	3.459		4	220	9.50	10.52
	14	14	86.09	1.162		22	118	28.30	3.534		6	222	9.30	10.75
	16	16	84.27	1.187	5	0	120	27.70	3.610		8	224	9.11	10.98
	18	18	82.48	1.212		2	122	27.11	3.688		10	226	8.91	11.22
	20	20	80.74	1.239		4	124	26.54	3.768		12	228	8.72	11.46
	22	22	79.03	1.265		6	126	25.98	3.849		14	230	8.54	11.71
1	0	24	77.36	1.293		8	128	25.43	3.933		16	232	8.36	11.96
	2	26	75.72	1.321		10	130	24.89	4.018		18	234	8.18	12.22
	4	28	74.12	1.349		12	132	24.36	4.105		20	236	8.01	12.49
	6	30	72.55	1.378		14	134	23.85	4.193		22	238	7.84	12.76
	8	32	71.01	1.408		16	136	23.34	4.284	10	0	240	7.67	13.03
	10	34	69.51	1.439		18	138	22.85	4.377		6	246	7.20	13.90
	12	36	68.04	1.470		20	140	22.36	4.471		12	252	6.75	14.82
	14	38	66.60	1.502		22	142	21.89	4.568		18	258	6.33	15.80
	16	40	65.19	1.534	6	0	144	21.43	4.667	11	0	264	5.94	16.85
	18	42	63.81	1.567		2	146	20.96	4.768		6	270	5.57	17.96
	20	44	62.46	1.601		4	148	20.53	4.871		12	276	5.22	19.16
	22	46	61.13	1.636		6	150	20.10	4.976		18	282	4.90	20.43
2	0	48	59.84	1.671		8	152	19.67	5.084	12	0	288	4.59	21.78
	2	50	58.57	1.707		10	154	19.25	5.194		6	294	4.31	23.22
	4	52	57.33	1.744		12	156	18.85	5.306		12	300	4.14	24.76
	6	54	56.12	1.782		14	158	18.45	5.421		18	306	3.79	26.41
	8	56	54.93	1.820		16	160	18.06	5.538	13	0	312	3.55	28.15
	10	58	53.77	1.860		18	162	17.67	5.658		6	318	3.33	30.03
	12	60	52.63	1.900		20	164	17.30	5.780		12	324	3.13	32.00
	14	62	51.52	1.941		22	166	16.93	5.905		18	330	2.93	34.13
	16	64	50.43	1.983	7	0	168	16.58	6.033	14	0	336	2.75	36.40
	18	66	49.36	2.026		2	170	16.22	6.164		6	342	2.58	38.81
	20	68	48.31	2.070		4	172	15.88	6.297		12	348	2.42	41.38
	22	70	47.29	2.115		6	174	15.55	6.433		18	354	2.27	44.13
3	0	72	46.29	2.160		8	176	15.22	6.572	15	0	360	2.13	47.05
	2	74	45.31	2.207		10	178	14.89	6.714		12	372	1.87	53.49
	4	76	44.35	2.255		12	180	14.58	6.859	16	0	384	1.64	60.83
	6	78	43.41	2.304		14	182	14.27	7.008		12	396	1.45	69.14
	8	80	42.49	2.353		16	184	13.97	7.159	17	0	408	1.27	78.63
	10	82	41.59	2.404		18	186	13.67	7.314		12	420	1.12	89.41
	12	84	40.71	2.456		20	188	13.38	7.472	18	0	432	0.984	101.7
	14	86	39.86	2.509		22	190	13.10	7.634		12	444	0.865	115.6
	16	88	39.01	2.564	8	0	192	12.82	7.800	19	0	456	0.761	131.4
	18	90	38.18	2.619		2	194	12.55	7.968	20	0	480	0.589	169.9

of the 411 kev. gamma rays emitted by Au^{198} is 0.225 cm.²/g. (ref. 36). From this the half-thickness in lead may be calculated to be $0.693/(0.225 \times 11.1) = 0.277$ cm.

The calculated mass absorption coefficient in soft tissue of the 0.411 mev. gamma rays is 0.100 cm.²/g. (ref. 36). If the density of the tissue be taken as 1.08, the calculated half-thickness in soft tissue is 6.41 cm.

Beta Particle Absorption. The ranges of the beta particles in aluminum and gold were determined by interposing foils of these metals between the thin mica window of a Geiger counter tube and a strong source of Au^{198} in the form of a drop of colloidal radioactive gold* evaporated on a very thin plastic film supported in such a way as to minimize backscatter, according to the method described by Glendenin.[11] The beta/gamma activity ratio, which was 15.5, was determined by extrapolation to zero aluminum absorber (Table VI). The maximum range in aluminum determined visually was 402 milligrams per square centimeter,[20] corresponding to a thickness of 1.5 mm. of aluminum. The experimental value agrees very well with the range of 393 mg./cm.² calculated for the range of a beta particle with energy of 0.97 mev. by means of Glendenin's equation based on experimental data for electrons and nuclear beta particles. The maximum range in gold determined visually was 442 mg./cm.², but the cut-off thickness was not sharp because the end-point was obscured by the gamma activity. This is in fair agreement with a range of 485 mg./cm.² calculated from Glendenin's value of 500 mg./cm.² in gold for a 1 mev. beta particle.

The range found here divided by the density for gold gives 0.23 mm. of gold required to stop all of the most energetic of the beta particles emitted by Au^{198}. But, because nuclear beta radiation has a continuous energy spectrum with the usual distribution approximately as shown in Figure 3, a gold-tubing beta-shield with wall 0.20 mm. thick was chosen since only an insignificant fraction of the beta particles will penetrate it and the energy of those which do pass through the shield will be so nearly spent that the damage they do to the surrounding tissues will not be excessive in comparison with that due to the desired gamma rays passing out through the beta shield (Table VI).

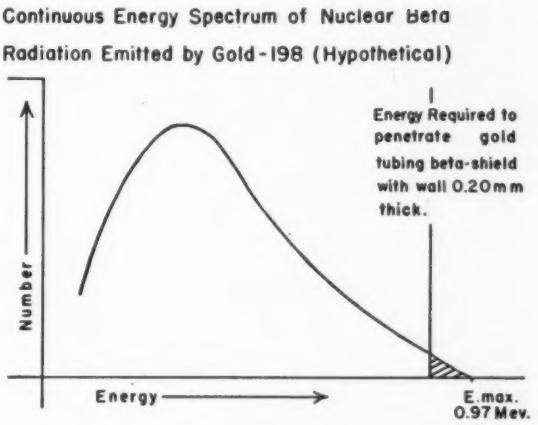
Continuous Energy Spectrum of Nuclear Beta Radiation Emitted by Gold-198 (Hypothetical)
Energy Required to penetrate gold tubing beta-shield with wall 0.20mm thick.
Number
Energy
E.max. 0.97 Mev.

FIG. 3. Continuous energy spectrum of nuclear beta radiation

SOURCES OF GOLD-198

Preparation. Radioactive gold sources suitable for deposition interstitially as seeds in the same way that radon seeds may be used, as well as to be incorporated in removable linear sources in radioactive plastic sutures[23,24] or stainless steel needles, or in molds, may be made easily and safely, and with very simple apparatus. The practice here has been to irradiate straight lengths of pure gold wire, about 8 cm. long and of suitable diameters, in the neutron flux of the nuclear reactor at the Oak Ridge National Laboratory for about a week. After the wires are received via air express in a nonreturnable shipping container, they are slid into gold tubing previously placed within a fairly close-fitting brass tube on the axis of a lead-shielded micrometer. The radioactive gold wire sources are pinched off within the nonradioactive gold beta-particle absorber with a pair of blunt-edged pliers* at the shield end as they are fed against the face of the micrometer, which is adjustable for length of cut.

Equipment. In Figure 4 are shown the simple

* Obtained from Abbott Laboratories, North Chicago, Illinois.

* Utica No. 1950 side-cutting pliers were found to be suitable for the purpose.[5]

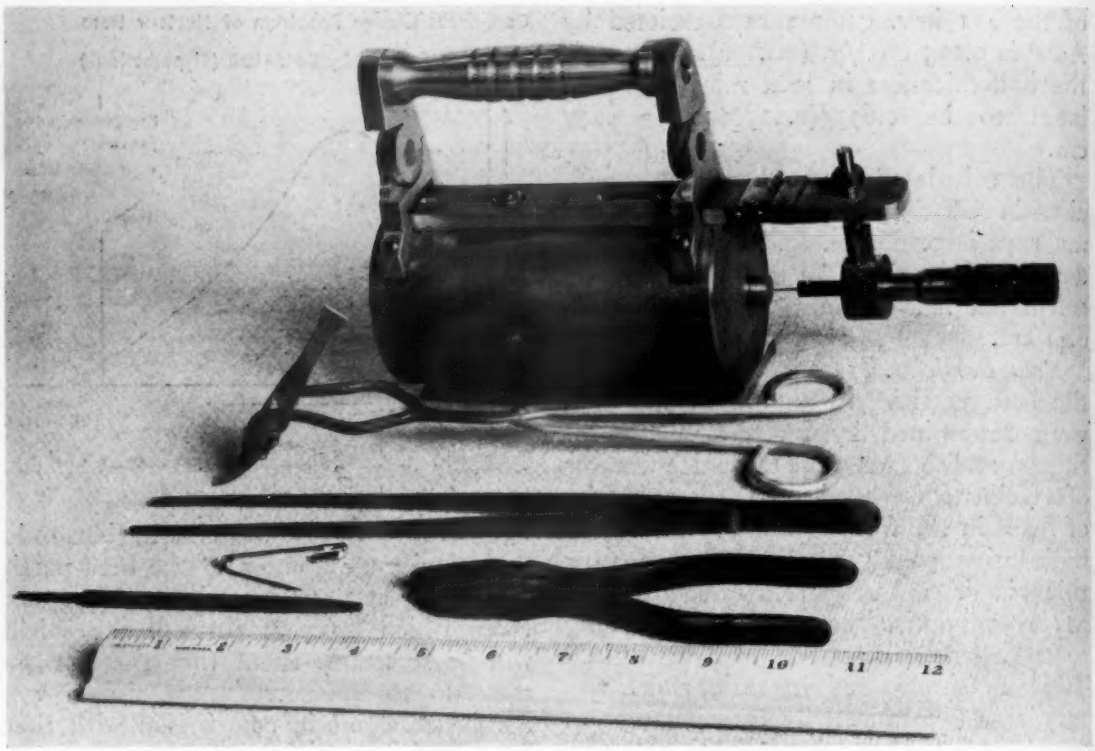

FIG. 4. Equipment used in the preparation of the gold-198 sources.

items of equipment used for making sources. The shielded micrometer,* which also serves as a storage shield when sources are not being prepared, is constructed of a piece of brass pipe about 6 inches long, 3.0 inches outside by 2.75 inches inside diameter, which is filled with lead, with a hole 0.5 inch in diameter drilled through the center. Inside this hole in the lead shield is fitted a brass cylinder $\frac{1}{2}$ inch outside diameter, with a hole $\frac{1}{16}$ inch in diameter on the axis. At one end of the larger shield cylinder there is mounted a micrometer with the center of the face aligned with the axis of the hole in the inner brass cylinder through which the gold tubing is pushed along with a piece of straight steel wire. The mount for the micrometer is hinged so that the plunger may be locked in line with the center hole of the inner cylinder during use, or it may be swung clear on the hinge.

Adjustability. By means of the micrometer the lengths of the seeds may be adjusted precisely, to fill the needs of individual radiotherapists and to allow for decay according to the reciprocals of die-out with time listed in Table 1. This adjustability of strengths on-the-spot is a feature of the method of preparing sources here which will be discussed later.

Several seeds of various lengths prepared routinely by the above method are shown in Figure 5. For comparison there is also shown a radon seed obtained from a commercial source.† It will be seen that this radon seed in the lower left corner of the photograph is almost indistinguishable in appearance from the gold-198 seeds prepared here. Longer sources to be placed in plastic sutures and stainless steel needles, or to be bent easily to fit molds, are also prepared in the same way. Views of the sources in cross-section are depicted‡ in Figure 6.

Loading and Protection. To facilitate loading, one end of the inert gold tubing is fanned out into a tiny funnel with the point of a safety pin before the other end is slid into the brass tube core at the end of the lead shield opposite to the micrometer mount. After a little practice with nonradioactive wire, one may transfer in less than twenty seconds a length of the irradiated

* Carl McWhirt, Director of the Physics Shop, and Earl Schofield, Director of the Medical Shop, assisted in the design and construction of the shielded micrometer.

† Kindly loaned by Professor Arthur G. James of the Department of Surgery.

‡ Prepared by Ralph P. Marinelli of the Medical Art Department.

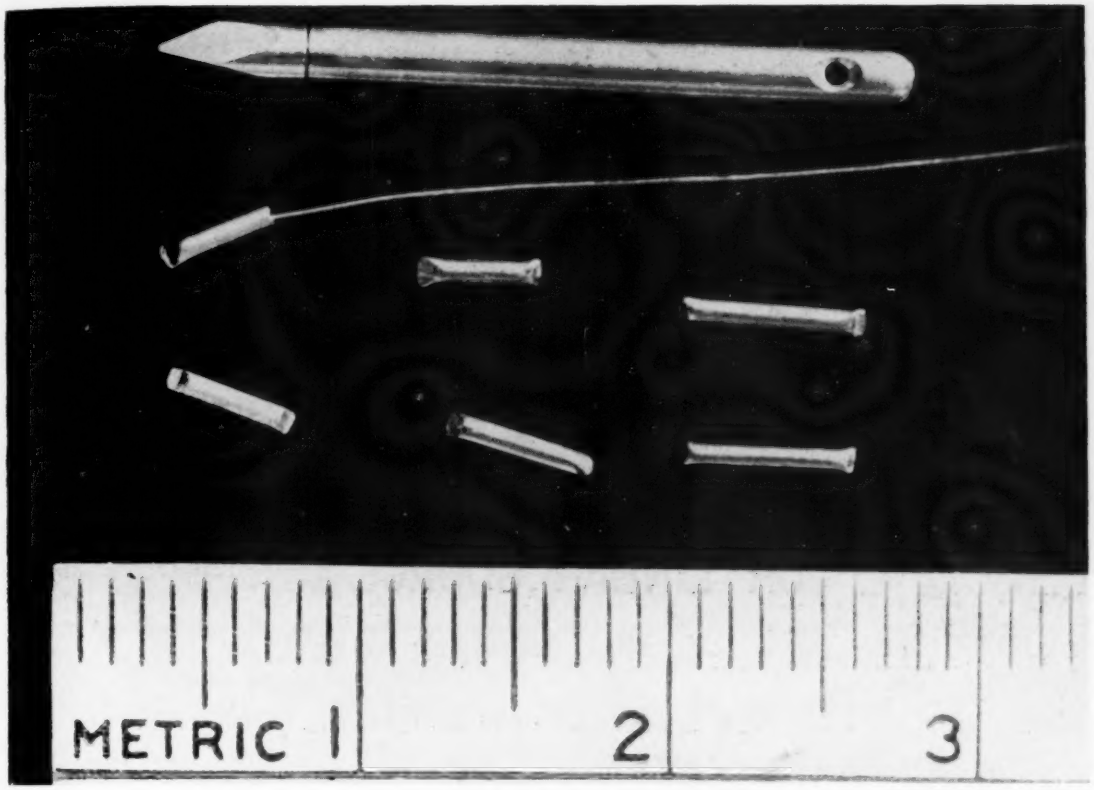

Fig. 5. Sources of gold-198 in fine gold wire enclosed in an inert gold-tubing beta-particle shield. A length of the gold wire is shown extending from an end of the gold tubing in one case. The seed at the lower left is a radon seed obtained from a commercial source. For comparison, a standard gold needle to hold one cell of radium is shown at the top.

wire from the aluminum exposure can in the shipping container shield in which it is received, into the inert gold tubing within the shield, with the 10 inch loading tongs shown in Figure 4. During this time very thick rubber gauntlets* and a plastic face shield 1.5 mm. thick are worn. The maximum exposure recorded by a pocket dosimeter strapped to the forehead in any one day of loading, when four lengths of radioactive gold wire were loaded and pinched off into seeds was only 18 milliroentgens. The lead in the shield wall is about 10 half-thicknesses for the 411 kev. gamma rays emitted by Au^{198}, so that the maximum rate of leakage with a full length of radioactive gold wire placed in the center of the shield soon after receipt from Oak Ridge is only 5 milliroentgens per hour.

Portability. The shielded micrometer weighs only 17 pounds and the whole apparatus may be kept loaded in the radioisotope laboratory with a length of the radioactive gold wire within the gold-tubing beta-particle shield, to be carried easily to any place in the hospital where sources of any desired strengths may be pinched off where they are to be used. This portability is due mainly to the fact that the half-thickness in lead of the 411 kv. gamma rays emitted by Au^{198} is only 0.28 cm. in contrast with the half-thickness of 1.39 cm. in lead[7] in the case of the gamma rays emitted by radium disintegration products.

Cost of Equipment. The costs for labor and materials to make the shielded micrometer were less than twenty-five dollars, which is a trifling sum in comparison with the capital investment in the apparatus and equipment used for preparing sources filled with radon.

STRENGTHS OF SOURCES

Units. Fortunately, in the same issue of the journal in which the announcement[1] of the

* These gauntlets have a thickness of 2.5 mm. of soft rubber and they absorb more than 95 per cent of the beta particles emitted by gold-198. They were manufactured especially for this purpose and kindly supplied by the B. F. Goodrich Company, Akron, Ohio.

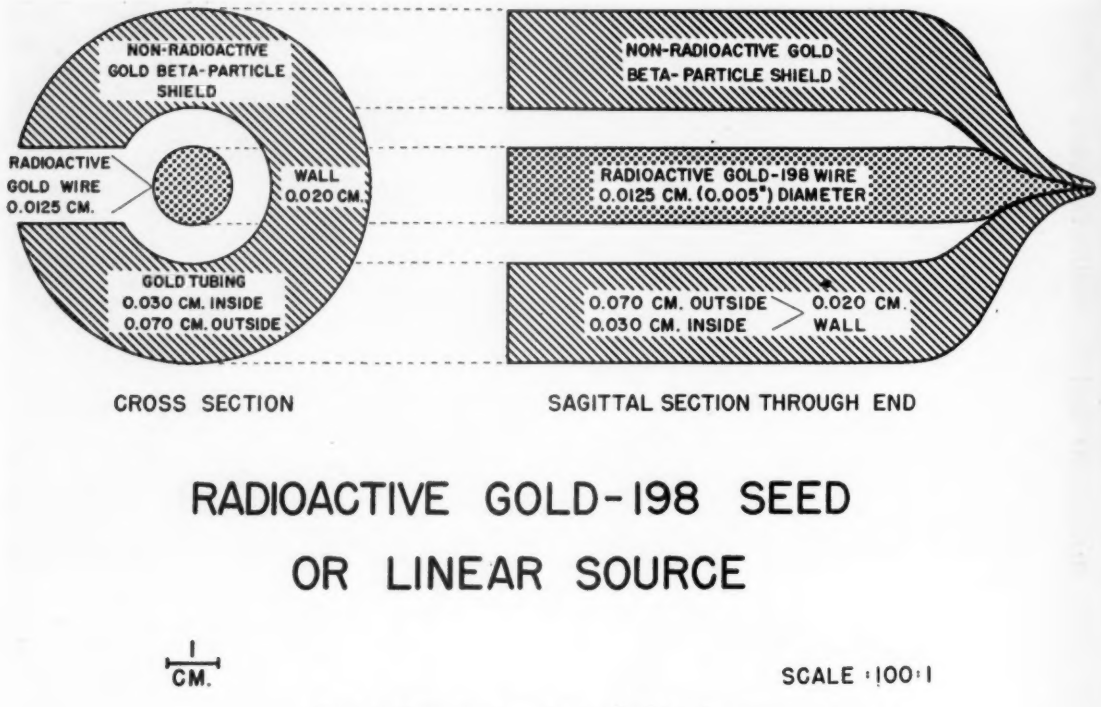
NON-RADIOACTIVE GOLD BETA-PARTICLE SHIELD
RADIOACTIVE GOLD WIRE 0.0125 CM.
WALL 0.020 CM.
GOLD TUBING 0.030 CM. INSIDE 0.070 CM. OUTSIDE
NON-RADIOACTIVE GOLD BETA-PARTICLE SHIELD
RADIOACTIVE GOLD-198 WIRE 0.0125 CM. (0.005") DIAMETER
0.070 CM. OUTSIDE
0.030 CM. INSIDE
0.020 CM. WALL
CROSS SECTION
SAGITTAL SECTION THROUGH END
RADIOACTIVE GOLD-198 SEED OR LINEAR SOURCE
1 CM.
SCALE 1:100:1

FIG. 6. Cross section views of gold-198 sources.

availability of large quantities of radioisotopes of interest in therapy appeared, there were proposed new units for the measurement of radioactivity.[6] One of these was the roentgen-per-hour at one meter, abbreviated r.h.m., which was introduced as a practicable unit in which the intensity from gamma-ray sources could be expressed conveniently without reference to the disintegration rate. Evans[9] first used a submultiple, the milliroentgen-per-hour at one meter, mrhm, in which the strengths of gamma-ray sources used in radiotherapy* may be measured easily with conventional equipment. Because radiotherapists have become accustomed to think of dosages in terms of the number of roentgens the irradiated tissues receive, it has been the practice here[3,25,26] to use the new unit for the evaluation of the strengths of sources of gamma rays emitted by the new radioisotopes under study.

Calibration of Instruments. Originally it had been planned to obtain from the National Bureau of Standards a source of gold-198, certified as to strength, for the calibration of measuring instruments but a suitable source was not available.[35]

A Victoreen condenser r meter with 250 milliroentgen chamber, which had recently been serviced and recalibrated by the manufacturer, was stated to have an accuracy of ±10 per cent, reproducibility ±3 per cent, with energy dependence less than 10 per cent from 50 kev. to 400 kev.;[4] and it was chosen as the basic instrument for the determination of the strengths of the radiogold sources. As a check of the manufacturer's specification the chamber was exposed to the gamma rays from a source of 9.54 mg. of radium in 0.5 mm. of platinum, certified by the National Bureau of Standards, which was supported on a nylon stocking stretched on a ring on a stand at various distances above the chamber. Excellent agreement was found between the experimental and the calculated values. The percentage differences ranged between 1.1 and 8.2 with an average of 4.1 per cent variation among 14 determinations.[20] A practicable distance between the sources and the center of the chamber was found to be 25.4 cm. (10 inches) consistent with reproducible readings over a large part of the scale with exposures lasting thirty to sixty minutes.

For many purposes it is desirable to have a reliable instrument available which has a higher sensitivity than the Victoreen 250 mr chamber and it was found that an inexpensive quartz

* One milligram of radium in radioactive equilibrium with its short-lived decay products in 0.5 mm. platinum gives rise to gamma radiation of 0.84 mrhm intensity.[9]

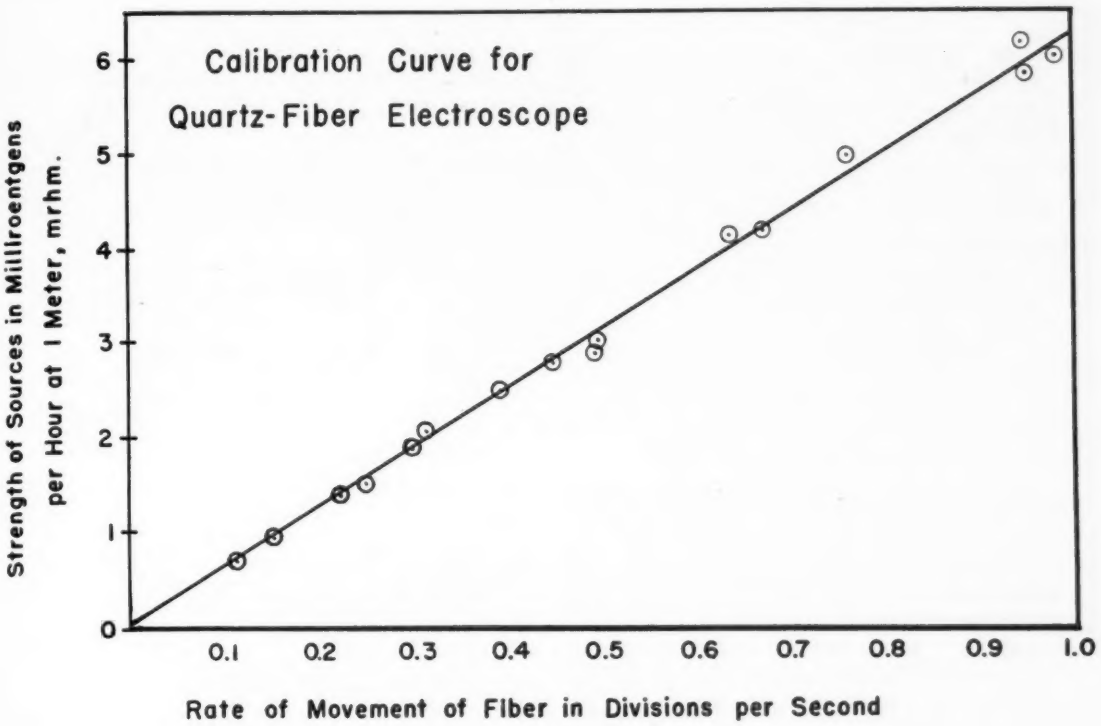

Fig. 7. Calibration curve of quartz fiber electroscope. The sources were 55 cm. above the center of the chamber.

fiber electroscope* gave quite satisfactory service. The practice has been to place the case of the instrument on the base of a ring stand and to turn the lid back on its hinge. The sources were supported on nylon stretched on a ring 55 cm. above the center of the sensitive volume of the electroscope. A plot of the rate of movement of the fiber as a function of the source strength determined with the Victoreen chamber is shown in Figure 7. It will be seen that a source with an activity of 2.5 mrhm caused the fiber to move at the rate of 24 divisions per minute.

Service Irradiations: Activity per Shipment. On the basis of measurements† at the surface of the shipping box containing a unit consisting of 7 mil gold wire irradiated in a flux of 44 per cent in the nuclear reactor at the Oak Ridge National Laboratory from May 5 to 12, 1952, it was found that the maximum amount of radioactive gold wire of this diameter which may be shipped in a nonreturnable container without exceeding 200 mr/hr. at the surface was about 200 mg. When this gold wire is enclosed in a gold-tubing beta shield with a wall 0.2 mm. thick, it gives rise to approximately 0.7 mrhm/mg. as it is shipped from the pile. The initial activity of the maximum amount of 7 mil gold wire which may be shipped in a nonreturnable container is therefore about 140 mrhm in the form in which it is commonly used here. This amount of activity corresponds initially to that emitted by approximately 170 millicuries of radon enclosed in 0.5 mm. of platinum. When this is multiplied by $2.7/3.825=0.70$ to take into consideration the differences in the lives of the radioisotopes, the amount of radiation emitted by the 200 mg. of 7 mil gold wire containing the Au^{198} during complete decay corresponds to that from about 120 millicuries of radon during lifetime decay.

Uniformity of Shipments. In Table II are shown the relative activities generated in 7 mil gold wire in the nuclear reactor at the Oak Ridge National Laboratory as determined in shipments received at various times and calculated on the basis of the time of removal from the pile, presumably at 8 A.M. on Monday of the

* Quartz Fiber Electroscope, Model 2, manufactured by Fred C. Henson Company, 3311 East Colorado Street, Pasadena, California.

† The cooperation of John H. Gillette, Superintendent of the Radioisotope Control Department of the Oak Ridge National Laboratory, in the making of these measurements is greatly appreciated.

TABLE II

UNIFORMITY OF ACTIVITIES GENERATED IN GOLD WIRE IN THE NUCLEAR REACTOR AT THE OAK RIDGE NATIONAL LABORATORY

Dates of Shipment	Activity in 7 mil Gold Wire mrhm*/cm.
2/18/52	3.50
3/31/52	3.88
4/ 7/52	3.72
4/14/52	3.37
	Average 3.62

* mrhm = milliroentgens per hour at 1 meter.

week the shipment was received. It will be seen that the range of variation in the activities was approximately 15 per cent.

Effect of Diameter of Wire on Relative Activity. In Table III are shown data collected from a recent shipment containing radioactive gold wires of the same length but of various diameters which had been mounted in aluminum foil inside the aluminum exposure can in such a way as to minimize "neutron shadowing" in the nuclear reactor. The decrease in relative activity per milligram with increase in diameter of the pieces was presumably due chiefly to the increase in the "self-shading" of the neutrons in the thicker wires because of the decreasing ratio of surface area/cross-section area, in conjunction with the high capture of slow neutrons by gold atoms.[30]

TABLE III

DECREASE IN RELATIVE ACTIVITY DUE TO NEUTRON SHADOWING

Diameters of Gold Wire*		Calculated Weight per cm.	Activity per Milligram Relative to That of 5 mil Gold Wire
mils	mm.†	mg.	
5	0.1270	2.45	1.00
6	0.1524	3.52	0.98
7	0.1778	4.79	0.94
8	0.2032	6.26	0.89
9	0.2286	7.92	0.88
10	0.2540	9.78	0.86
11	0.2794	11.83	0.82
12	0.3048	14.08	0.80
15	0.3810	22.00	0.75

* The "commercial tolerances" of the small diameter wires are believed to be ±0.0005 cm. according to a letter of February 6, 1952, to Wm. G. Myers from J. E. Krantz of The American Platinum Works, Newark, New Jersey.

† Calculated on the basis that 1 mil = 0.001 inch = 0.0254 millimeter.

TABLE IV

UNIFORMITY IN ACTIVITY OF GOLD-198 SOURCES

Au^{198} Wire	Source Length	Activity	Difference from Mean
mils	cm.	Counts / Second	Per Cent
6	1	51.5	−0.96
6	1	52.0	0.00
6	1	52.2	+0.38
6	1	52.7	+1.35
6	1	51.3	−1.35
6	1	51.1	−1.73
6	1	53.3	+2.50
		Mean 52.0	

Uniformity of Radiogold Sources. In Table IV are shown data taken with a Geiger counter on 7 sources, cut 1 cm. long, containing 6 mil radioactive gold wire from a recent shipment. The maximum variation from the mean was 2.5 per cent and the range was 4.3 per cent. When the activities were determined of several other lots of sources prepared with wires 4 to 7 mils in diameter, the maximum variation from the mean was 3.2 per cent.

TABLE V

VARIATION IN ACTIVITY OF RADON SOURCES

Radon Seed	Counts / Second	Per Cent Variation from Mean
1	165.9	+21.8
2	163.8	+20.3
3	124.8	− 8.4
4	167.1	+22.7
5	136.3	—
6	118.1	−13.3
7	126.4	− 7.2
8	127.8	− 6.2
9	121.5	−10.8
10	125.7	− 7.7
11	129.6	− 4.8
12	127.8	− 6.2
	Mean 136.2	

Table VI

BETA PARTICLE LEAKAGE

Sources	Range in Aluminum mg./cm.²	Maximum Energy in Mev.[11]	Maximum Range in Tissue in mm.[29]	Beta / Gamma at Zero Aluminum
A. Gold 198 seed (paraffin on ends)	90	0.33	1	0.5
B. Gold 198 seed (ends bare)	200	0.58	2	1.9
C. Gold 198 sol (colloidal, dried)	402	0.98	4	15.5
D. Radon seed	900	1.9	9	1.9

Variation in Radon Seeds. For comparison, the variations encountered in a shipment of a dozen radon seeds* with an average activity of 1.46 millicuries, obtained from a commercial source, are shown in Table v. The maximum variation from the mean was found to be 22.7 per cent and the range was 41.5 per cent.

The much greater uniformity of activity of the gold-198 sources prepared here over that found in the radon seeds is obvious.

BETA PARTICLE LEAKAGE BY RADON AND GOLD-198 SOURCES

Autoradiographs of the radon seeds and of some of the typical gold-198 sources showed evidence of leakages of beta particles at the ends. Absorption studies in aluminum foils were then made to determine the relative energies of the beta particles passing out of the sources. The results are tabulated in Table vi.

Source A was 1 cm. of the gold tubing with 0.2 mm. wall, enclosing 7 mil radioactive gold wire, and with the pinched-off ends covered with paraffin so that the leakage of beta particles occurred only through the wall. Probably some of the slight activity shown in Table vi was due to Compton electrons scattered from near the surface of the tubing.

Source B was the same as A before the ends were covered with the paraffin. The greater range of the beta particles clearly shows that some leakage occurred at the thinned part of the pinched-off ends of this source (Fig. 6). The autoradiographs also showed this leakage. The maximum range of only 2 mm. in soft tissue indicates that the leakage is of little significance since periacicular necrosis inevitably occurs in this region anyhow due to the high gamma-ray dosages received in the first few millimeters immediately adjacent to the sources.

The data for Source C are included in Table vi for completeness. They were considered above under the physical properties of gold-198.

Source D was one of the radon seeds obtained from a commercial supplier.† It will be evident that the maximum range of the beta particles from the radon seed was 4.5 times as great in soft tissue as that found in the case of the gold-198 source. Again, some of the electrons may have been Compton recoils.[31]

The maximum energy of the beta particles emitted by radium C in the chain of disintegration products of radon is 3.17 mev.[19,32] The range of electrons of this energy in aluminum is 1,590 mg./cm². (ref. 11). Data were not found in this energy region for the correction necessary to take into consideration the relative stopping power of gold and of aluminum due to the changing proportions of neutrons and protons in these elements. Assuming that it be proportional to Z/A (ref. 11), the correction will be $(13/27)/(79/197)=1.2$. The minimum thickness of gold required to stop all of the 3.17 mev. beta particles then will be $1.2\times1,590=1,900$ mg./cm². When this is divided by the density of gold, the thickness of gold required is 0.98 mm. The fact that the *diameter* of the gold-filtered radon seed was less than 0.8 mm. may account largely for the presence of electrons with a range in aluminum equivalent to 9 mm. in soft tissue.

* Kindly loaned by Professor Arthur G. James of the Department of Surgery.

† Kindly loaned by Professor Arthur G. James of the Department of Surgery.

TABLE VII

APPROXIMATE STRENGTHS OF SOURCES OF GOLD-198
(Based on Victoreen 250 mr Chamber)

At 8 A.M. each day after removal from the nuclear reactor at the Oak Ridge National Laboratory, and after enclosure of the radioactive gold wires in gold tubing with wall 8 mils thick

	A mrhm*/cm.			B Total r/4 mm. at 1 cm. for lifetime decay			C Lifetime decay/4 mm. / Lifetime decay of 1 mc. of radon		
Mils of Au Wire	7	10	15	7	10	15	7	10	15
Monday	3.62**	6.73***	13.29***	1350	2520	4970	1.21	2.26	4.47
Tuesday	2.80	5.21	10.29	1050	1950	3850	0.93	1.75	3.46
Wednesday	2.16	4.03	7.95	810	1510	2970	0.73	1.35	2.67
Thursday	1.68	3.12	6.15	630	1170	2300	0.56	1.04	2.07
Friday	1.30	2.41	4.76	480	900	1780	0.44	0.81	1.60
Saturday	1.00	1.86	3.68	380	700	1380	0.34	0.63	1.24
Sunday	0.77	1.44	2.84	290	540	1060	0.26	0.48	0.95
Monday	0.60	1.12	2.21	220	420	820	0.20	0.38	0.74
Tuesday	0.46	0.86	1.69	170	320	630	0.16	0.29	0.57

* mrhm = milliroentgens per hour at 1 meter.
** Average value for 4 shipments.
*** Corrected for variations in relative activity with diameter of wire.

ACTIVITIES OF SOURCES FOR THERAPY

Table VII shows approximate activities of sources calculated for the days of the week (Table I) and based on the average value of 3.62 mrhm/cm. shown in Table II for the activities generated in 7 mil gold wire in four different irradiations. The values for the 10 mil and 15 mil wires were corrected in accordance with Table III.

For the preparation of linear sources to incorporate in removable plastic sutures or stainless tubing, or in molds, the values calculated in terms of mrhm/cm. in part A of Table VII should be useful.

In part B are shown the approximate values of the total number of roentgens that will be received at a point 1 cm. from a seed 4 mm. long during the complete decay of the activity in the seed. An example will illustrate how the values in this part of the table were calculated:

$$3.62 \text{ mrhm/cm.} \times 0.4 \text{ cm.} \times 0.463 \text{ of original activity} \times 93.5 \text{ hours average life} \times \left(\frac{15}{7}\right)^2 \times \frac{0.75}{0.94} \times \frac{100^2 \text{ mr/hr. at 1 cm.}}{1000 \text{ mr/r}} = 2{,}300 \text{ r}$$

during lifetime decay at 1 cm. from a seed 4 mm. long prepared from 15 mil radioactive gold wire at 8 A.M. on Thursday.

Part C gives the relationship between the total number of roentgens received at a point 1 cm. from gold-198 seeds 4 mm. long during complete decay as compared with the 1,112 r which would be received at the point if the radioactive gold seed were replaced by a seed containing 1 millicurie of radon enclosed in 0.5 mm. of platinum. For the two seeds implanted at 8 A.M. on Thursday this ratio would be 2,300/1,112 = 2.07.

This part of Table VII should be especially useful for radiotherapists who will wish to use seeds containing Au^{198} in place of the radon seeds they may have been using. Should they want the gold-198 seeds to give off radiation equivalent to that from radon seeds containing 1 millicurie each during lifetime decay, they would make

them of 7 mil gold wire with activity of 2.80 mrhm/cm. at 8 A.M. on Tuesday and cut to lengths of $4/0.94=4.3$ mm. On Wednesday they would use the 10 mil radiogold wire and cut the seeds $4/1.35=3.0$ mm. long. On Thursday they would use the same wire but the seeds would be cut $4/1.04=3.8$ mm. long. By Friday they would probably elect to use the 15 mil wire and the seeds would be $4/1.60=2.5$ mm. long. For Saturday the 15 mil radioactive gold wire would be used and the seeds cut $4/1.24=3.2$ mm. long.

It will be obvious from the examples above how to make the appropriate adjustments for wires in which the initial activities vary from 3.62 mrhm/cm. for 7 mil wire; for times of implantation other than 8 A.M. on any day; and for seeds with total radiation activities of strengths greater or lesser than that equivalent to the amount given off by 1 millicurie of radon. Thus, a single shipping container received from the Oak Ridge National Laboratory on Monday evening by air express might contain radioactive gold wires of various diameters and activities so that seeds of any desired strengths and about 3–4 mm. long may be prepared from it to fulfill any need that may arise during a whole week. This on-the-spot adjustability of gold-198 sources is a feature of Au^{198} which cannot be matched by radon except in a very few large centers where radon plants are available.

COST OF GOLD-198 SEEDS

An estimation of the approximate cost for the Au^{198} seeds may be useful. If one assumes the seeds will be used at 8 A.M. on Wednesday and that each of them should have an activity such that a point 1 cm. from the seed will receive 1,112 roentgens during complete decay, *i.e.*, the amount of radiation the point would receive from 1 millicurie of radon during lifetime decay in a platinum seed with 0.5 mm. wall, then the radioactive gold wire in the seed will be 10 mils in diameter and 3 mm. long (Table VII). About 70 of these seeds could be prepared on Wednesday morning from the maximum amount of the radioactive gold which could be shipped from the Oak Ridge National Laboratory in a nonreturnable container. The approximate costs would be:

For gold wire and gold tubing	$ 4.00
Service irradiation for one week	12.00
Handling charges	10.00
Air express—varies with distance	~4.00
Labor—for cutting and measurements	5.00
Total	$35.00

The cost for each of the above seeds will then be about one-half dollar, including labor costs.

For cancer treatment centers where there may be need for very large quantities of radiogold, and suitable procurement and shipping arrangements could be negotiated, the cost of the seeds might presumably be reduced to about one-fourth of the above estimate.[8]

DISCUSSION

The ideal time to implant radioactive seeds in a tumor is usually at the time the decision is made to use them; especially is this true when a cancer that cannot be excised completely is encountered unexpectedly during surgery, and it would be most desirable to have the seeds available within a very short time to insert into the tumor. The methods described here for the preparation of radioactive seeds containing gold 198 indicate that it is feasible and easy to have seeds of any desired strengths available for implantation at any time a need for them may arise. The apparatus required is simple in design and inexpensive to make; and there is no capital investment comparable with that required to establish and to maintain a plant for preparing seeds loaded with radon.

The data presented, which show the properties and characteristics of the radiogold seeds, clearly indicate their superiority in comparison with radon seeds, particularly in regard to uniformity of strengths (Table IV). Another point for consideration

is the fact that Au^{198} decays completely and leaves no residual radioactivity. Radon, on the other hand, is the first member of a long chain of disintegration products one of which, radium D, has a half-life of 22 years and emits several gamma rays with energies up to 47 kev. Thus, radon seeds that are implanted to be left permanently continue to give off radiation with a half-thickness in soft tissues of about 1 inch during all of the time that they remain in the body.

The half-life of 2.7 days for gold-198 may appear at first to be a disadvantage. The difference between it and the half-life of 3.825 days for radon is not sufficiently great to cause concern on the part of those who are familiar with the use of radon and have become accustomed to dealing with radioactive sources with half-lives of a few days. Serial roentgenograms often show considerable migrations of interstitial sources to have occurred during rather short times, so that the shorter half-life of Au^{198} may actually be advantageous insofar as a proportionately higher amount of the intended dose of radiation is delivered before the seeds have moved so far from the sites of implantation.

Some of the other advantages of sources of gold-198 for interstitial therapy over those containing radon, radium, or cobalt 60 were outlined recently.[27] Among these is the fact that sources of radiogold are in the form of solids so that they are not subject to loss of active material in processing. Radon sources may leak gas and become relatively inactive. Moreover, leakages of the highly radioactive gas encountered in the maintenance of a radon plant and in the preparation of radon seeds make the handling of radon a hazardous occupation. And there is ever present the possibility of breakage of the vessel containing the solution of radium, or of spillage and contamination due to inadvertent unfavorable changes in pressure during the pumping procedures. Risks of inhalation of a dangerous radioactive gas are eliminated when sources of gold-198 are handled or processed.

The chemical inertness of gold is well known and, when radioactive forms of the element are used, there is no hazard of dealing with a bone-seeking long-lived radioactive powder as in the case of radium and no risk of troublesome contamination of facilities with long-lived radioactive flakes and dust due to the "rusting" encountered on the surfaces of some forms of radiocobalt, especially pure cobalt cylinders containing Co^{60}. Any contamination due to Au^{198} which might conceivably occur will be self cleansing rapidly with time in the sense that only about 10 per cent will remain on the ninth day, about 1 per cent on the eighteenth day, etc. (Table 1). A whole curie of Au^{198} in a room will have decayed to less than 3.5 disintegrations per second at the end of three months. With radium, the problem of permanent contamination is ever present and constant vigilance is required to avoid radium spills and accidents;[12] whereas any gold-198 that may become lost quickly becomes innocuous because of decay.

The 0.411 mev. gamma rays emitted by gold-198 are monoenergetic, and this homogeneity simplifies calculations of doses in comparison with radium and radon, which emit a dozen different photons with energies between 0.184 mev. and 2.198 mev.[9]

The strengths of the Au^{198} seeds, *prepared where they are to be used*, are easily maintained constant at any desired value either by (a) pinching them off slightly longer successively in inverse proportion to the die-out (Table 1), or (b) pinching them off with essentially similar lengths, after inserting radioactive gold wires of appropriately increasing diameters to compensate for the die-out (Table VII), into the gold-tubing beta-particle shield. This adjustability, *on-the-spot*, of sources with essentially unchanged physical dimensions, so they possess that strength deemed most appropriate by the radiotherapist, is a

distinct advantage because it eliminates a frequent difficult variable in the simultaneous matching of the availability of the patient, the hospital bed, the radioactive seeds, and the time of the therapist.

It should prove to be practicable and feasible in many circumstances, and especially in cancer hospitals, to stock the radioactive gold wires in various diameters and linear strengths in the shielded micrometer in the operating room area. The seeds may then be prepared in any desired activities within minutes of the time the therapist encounters a lesion in which he wishes to place them. Because the gold tubing is inert to almost all chemical reagents, the sources may be sterilized by immersion for a few minutes in strong acids, bases, or other suitable chemical solutions. Or, since there is no radioactive gas to be dealt with, they may be heated to very high temperatures in a few minutes in a crucible nesting on a small electric coil, or by induction heating.

Sources of Au^{198} should prove to be much more versatile even than those containing Co^{60}. For the seeds not only have the advantage of being applicable for implantation permanently interstitially, a way in which Co^{60} cannot be used because of its long life; but they can also be placed in plastic tubing to make flexible removable radioactive sutures which the surgeon may sew into cancer that he cannot remove completely, in exactly the same manner as the therapist may now use sutures loaded with Cobalt-60[17,18,23,24] as an adjunct to surgery for cancers which involve vital structures. And Au^{198} may be placed as seeds in place of cylinders containing Co^{60} within hollow stainless tubing to form removable needles for interstitial use. The gold tubing containing the radioactive gold wire may be bent easily to shape to incorporate into molds. Tests are now being carried out here to determine the feasibility of placing the radioactive gold wire directly in stainless tubing beta-shields with wall thickness of about 0.5 mm. to be made into needles to be used similarly to Co^{60} sources.[2,22,25,26]

SUMMARY

1. Radioactive gold, Au^{198}, with half-life of 2.7 days may be generated readily in pure gold wire of small diameter in a nuclear reactor.

2. The radioactive gold wire is slid into a nonradioactive gold tube with wall 0.2 millimeter thick which absorbs almost all of the 0.97 mev. (max.) beta particles and passes about 90 per cent of the 411 kev. gamma rays.

3. An easily constructed and inexpensive portable shielded micrometer and other simple items of equipment are described by means of which radioactive seeds and linear sources may be pinched off uniformly, quickly, and safely.

4. The radioactive gold seeds are suitable to be implanted permanently interstitially; and the linear sources may be incorporated in plastic tubing to make radioactive sutures, in stainless tubing to make radioactive needles, and they may be bent readily to shape into molds.

5. The strengths of the gold-198 sources may be adjusted at any time *at the point of usage* to suit any need of the radiotherapist by increasing the length of cut to allow for decay, or by choosing radioactive gold wire of suitable diameter. In contrast, radon sources are received from the manufacturer with the average strength specified for a given time and the strength may not be altered after receipt.

6. The strengths of the radiogold sources are quite uniform. For one lot of seven sources 1 cm. long the maximum variation from the mean strength was 2.5 per cent and the range was 4.3 per cent. Among a dozen radon seeds the maximum variation from the mean was found to be 22.7 per cent and the range was 41.5 per cent.

7. Among other advantages of the gold-198 sources over radon are: Inconsequential leakage of beta particles through the gold shield; no hazardous radioactive gas

to leak and no long-lived radioactive solution involved in the preparation to spill and produce permanent contamination; no capital investment in radium and in pumping equipment; decays completely and leaves no long-lived residual activity to cause low grade chronic radiation exposure.

8. The gamma rays emitted by gold-198 are monoenergetic in contrast with the heterogeneity of the gamma photons emitted from sources containing radium and radon.

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DOSIMETRIC CONSIDERATIONS IN DETERMINING HEMATOPOIETIC DAMAGE FROM RADIOACTIVE IODINE[1,2,3]

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THERE is no specific information that permits more than a rough approximation of the amount of I^{131} which in the human will cause severe but non-lethal damage and there is none that permits any calculations of the fatal dose. One pertinent item is found in the case reported from this institution of a patient who died subsequent to several large doses of I^{131}. The cumulative dose of I^{131} this patient received was 638 millicuries, and her death appeared to be the result of a pancytopenia. Other data on patients who received large doses of I^{131} without a fatal consequence show that the main toxic effect appears to be exerted on the blood forming organs. This is not surprising for as long ago as 1904 Heineke[4] noted that in animals roentgen rays caused the most serious damage to blood forming organs. From a practical standpoint it would be of considerable help if some idea could be obtained of the maximum safe dose of I^{131} for humans. Unfortunately several cases seen in the last few years at Memorial Hospital have suggested that a simple dose response curve in which the dose is measured in millicuries could probably not be obtained. The present study was undertaken, therefore, to attempt an answer to the following questions:

(1) What is the most reliable and sensitive indication of radiation damage from I^{131}?

(2) What is the most accurate means of determining the radiation actually delivered by any large dose of I^{131}?

(3) Can the amount of radiation delivered by any large dose of I^{131} be predicted by a prior tracer study?

METHODS

All radioiodine was received from Oak Ridge and standardized at either Memorial Center or Brookhaven National Laboratory. All patients were given I^{131} orally to which 2 to 10 micrograms (μg.) of I^{127} had been added as carrier. The urine was assayed for radioactivity by means of a Marinelli cup counter. The blood (whole blood was used exclusively) was assayed in a Texaco gamma-ray tube, and *in vivo* measurements were made with a Texaco gamma-ray tube. These methods have been described previously.[5] Blood counts were done in the usual manner and were taken daily for at least thirty days after any large dose of I^{131}. A running, unweighted, 3-point average was made of the neutrophils and lymphocytes after each large dose of I^{131} and per cent fall from the mean of at least three pretreatment levels was calculated from the lowest value thus obtained. In some instances the first blood measured for I^{131} was withdrawn twenty-four hours after the administration of the isotope. The concentration of I^{131} in the blood prior to the first determination was assumed to be equal to that found at that time. This is obviously incorrect and the magnitude of this error will be discussed later. The level of I^{131} in the blood was determined daily for at least twenty-five days and the value at the time of the last specimen extrapolated to infinity.

CALCULATIONS

The radiation the blood forming organs received from the blood was calculated as follows. Complete absorption of the beta rays was as-

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[3] This paper was presented in part before the meeting of the American Goiter Association in May, 1952.

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sumed. The radiation contributed by the gamma rays was ignored. The equation derived by Marinelli is:

$$\text{rep } \beta = 88 \times \text{average energy } \beta \text{ rays} \times \text{half-life (days)} \times 0.083 \times \text{microcurie days/gm.} \quad (1)$$

The mean beta-ray energy has been determined and also calculated by Marinelli and his coworkers. However, more recent data[6,12] show that I^{131} disintegrates with the emission of two beta rays of energies 0.600 mev. (85 per cent) and 0.315 mev. (15 per cent). One may calculate the average energy of these beta rays from the Fermi equations of allowable transitions by beta-ray decay for isotopes of a given Z. The average energies of the two beta rays then become 0.189 mev. and 0.0945 mev. Substituting in the above equation we have:

$$\text{rep } \beta = 10.2 \times \text{microcurie days/gram} \quad (2)$$

Millicurie days may be determined directly by planimeter of the microcuries per gram of blood.

The integrated dose of I^{131} retained by the patient was plotted as the difference between the amount administered and that excreted in the urine. The amount in the urine was measured every twenty-four hours for at least twenty days. Integration of the I^{131} retained was done with a planimeter. The amount of radiation that the blood received from I^{131} concentrated in any area in the body was calculated as follows.

It is assumed that all beta rays from the I^{131} in tumor or thyroid are absorbed in the tumor or thyroid and do not contribute to the radiation the blood forming organs receive. The body is assumed to be a cylinder of dimensions height 60 cm., radius 20 cm. The entire amount of energy emitted by the gamma radiation is given by Equation 3.

$$R_\gamma = E_\gamma \cdot 3.7 \cdot 10^4 \cdot c/\lambda, \quad (3)$$

where

R_γ = energy emitted,
E_γ = energy of all γ rays (in mev.),
c = microcuries destroyed per gram,
λ = decay constant (sec.$^{-1}$),
D_γ = mean radiation received by volume V.

When $W = 32.3$ e.v. = energy required to produce 1 ion pair in air and $N = 1.62 \times 10^2$ = number of ion pairs formed per gram of air by 1 roentgen, then:

$$D_\gamma = cE_\gamma \ (3.7 \times 10^4) \left(\frac{1}{\lambda \times W \times N \times V} \right) \quad (4)$$

(V = volume of cylinder in cm.^{3})

$D_\gamma = 3.81 \times 10^{-3}$ r/μc. destroyed

or,

$$D_\gamma = 0.316 \text{ r/millicurie day.} \quad (5)$$

This represents the average radiation received by the cylinder if all the gamma-ray energy is absorbed. From the half-value thickness for gamma rays of 0.605 mev. and 0.364 mev. in tissue it would appear that about 90 per cent of the energy is absorbed. Of course much of the energy is absorbed in the first 5 cm. or so of tissue and does not reach the blood forming organs. One would expect therefore that a more accurate value of the radiation would be half, or even less, the calculated amount. Certainly the most these calculations afford is an approximation of a mean value for radiation the blood forming organs receive.* The values found can in no way be compared to the roentgen or the roentgen equivalent in absolute terms. The radiation calculated in this manner will be referred to as "body retention" radiation. Body retention factors of 0.09, 0.08, etc., refer to radiation calculated in the same manner except for the use of different constants which are indicated by the figure used.

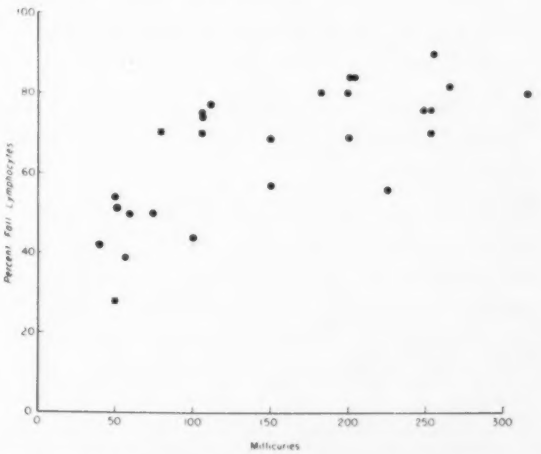

FIG. 1. Maximal per cent fall from pretreatment level in total circulating lymphocytes. 1. Millicuries of I^{131} administered.

* It may be noted that the roentgen equivalent physical (rep) as calculated here differs from the rep as calculated by Marinelli and Hill[10] which we used previously.[15] The principal differences are in ignoring the gamma-ray contribution from the blood and including a gamma-ray contribution from the I^{131} retained in the tumor. In general the rep as herein calculated gives somewhat smaller values than were previously reported.

RESULTS

Figure 1 shows the results of a plot of the millicuries of I^{131} administered to 27 patients plotted against the maximal per cent fall from pretreatment level in total circulating lymphocytes. It is apparent that although there is a greater fall in lymphocytes with a greater dose, no satisfactory correlation is obtained. Figure 2 shows the result of a plot of the radiation received by the blood calculated as previously described (Equation 2) versus the per cent

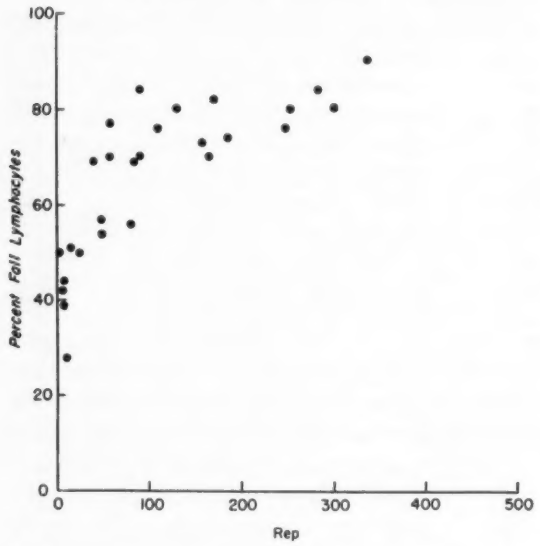

Fig. 2. Maximal per cent fall from pretreatment level in total circulating lymphocytes. 2. Radiation received by the blood.

fall from pretreatment level in total circulating lymphocytes. It appears that the correlation is considerably improved. It might be noted that the constants used in calculating the blood rep are unimportant from the standpoint of intragroup comparison as they will only rotate the coordinates about the XY intercept and will not influence the basic correlation observed. Figure 3 shows the result of a plot of the radiation received by the blood for 27 patients versus the maximal per cent fall from pretreatment level in total circulating neutrophils. It can be seen that the correlation obtained is considerably less satisfactory than if the same blood radiation calculations are plotted against the fall in

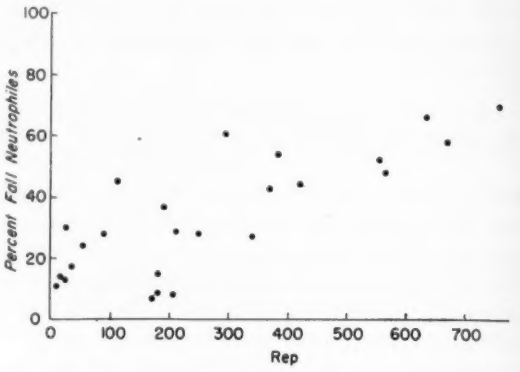

Fig. 3. Maximal per cent fall from pretreatment level in total circulating neutrophils. 1. Radiation received by the blood.

lymphocytes. Figure 4 shows the correlation of the radiation received by the blood forming organs as calculated by the blood radiation plus the previously described body retention factor (Equation 5). Instead of using the constant 0.316 rep/millicurie day, however, quite arbitrarily a constant of 0.090 rep/millicurie day has been used. In Table 1 are shown the equations derived by the method of least squares for each of these graphs assuming the general form of the equation to be $Y=A+B$

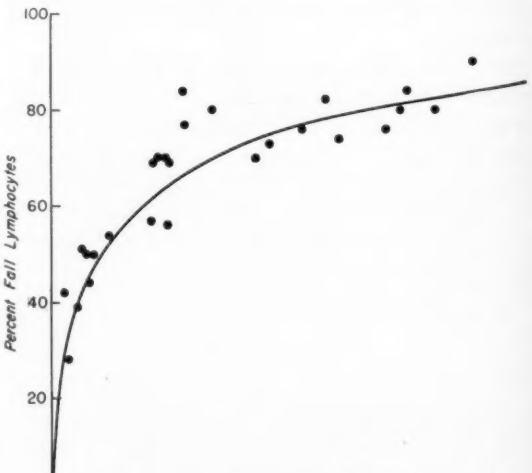

Fig. 4. Maximal per cent fall from pretreatment level in total circulating lymphocytes. Radiation received by the blood forming organs, as calculated by the blood radiation plus body retention factor 0.090.

TABLE I

CORRELATION OF MAXIMAL PER CENT FALL FROM PRETREATMENT LEVELS IN CIRCULATING LYMPHOCYTES IN 27 PATIENTS GIVEN A LARGE DOSE OF I^{131} WITH VARIOUS WAYS OF CALCULATING THE RADIATION AND CORRELATED BY SEVERAL DIFFERENT EQUATIONS

Y = Lymphocyte Fall
X = Radiation

Factor	Equation	Standard Error of Prediction	Correlation Coefficient	Probable Error of C. C.
Blood rep	$Y=14.5+28.7 \log X$	±7.9	0.867	0.032
Body retention rep	$Y=-24.0+42.3 \log X$	±9.1	0.627	0.078
Blood+0.09 body retention rep	$Y=-11.4+37.2 \log X$	±6.3	0.919	0.020
Blood+0.15 body retention rep	$Y=-18.0+38.4 \log X$	±6.6	0.909	0.022
Blood+0.09 body retention rep	$Y=\frac{X}{.324+.0117X}$	±6.7	0.906	0.023
Blood+0.15 body retention rep	$Y=\frac{X}{.431\pm.0117X}$	±6.9	0.900	0.025
Blood+0.09 body retention rep	$Y=100\ (1-0.59e^{-.00070X})$	±0.12	0.834	0.039
Blood+0.15 body retention rep	$Y=100\ (1-0.60e^{-.00058X})$	±0.12	0.827	0.041

log X. In these equations Y = lymphocyte fall and X = radiation. It can be seen from the correlation coefficients and the standard errors of prediction that the maximal per cent fall from pretreatment level in total circulating lymphocytes is best correlated by assuming that it is a function of the integrated concentration of I^{131} in the blood plus an arbitrary factor taking into account the radiation emitted by I^{131} retained in the body. The equations and correlation coefficients are based on the assumption that both X and Y (radiation and lymphocyte depression) are equally subject to error. That is, the constants derived from assuming X to be without error and Y to be without error are averaged. Several other equations were examined to see which gave the best fit to the experimental values. The equation $Y=X/(A+BX)$ when fitted to the points obtained using the blood rep plus the 0.09 body retention factor gave a correlation coefficient (0.906) almost identical with that using the equation $Y=A+B \log X$. With this equation, too, the best fit was obtained by correlating the lymphocyte fall with the blood radiation plus the 0.09 body retention factor. Similarly the data fit the equation $Y=Y_0(1-Ae^{-\lambda X})$ with a reasonably good correlation (correlation coefficient 0.834). Of particular interest is that with all three equations the best fit is achieved when Y = lymphocyte fall and X = blood rep+0.09 body retention factor.

These data so far suggest that with any given large dose of I^{131} the response of the hematopoietic system is reasonably well correlated with the radiation received by the hematopoietic system as calculated above. It furthermore demonstrates that the radiation the hematopoietic system receives from any given dose of I^{131} is a function of much more than the I^{131} administered to the patient and is to a very large measure a function of the manner in which that particular organism metabolizes iodine.

It became a matter of interest to see

TABLE II

CORRELATION OF THE PER CENT OF ADMINISTERED DOSE EXCRETED IN THE URINE IN 48 HOURS AFTER A TRACER AND AFTER A THERAPEUTIC DOSE OF I^{131} IN 27 PATIENTS. THE AVERAGE THERAPEUTIC DOSE WAS 151 MILLICURIES AND THEY RANGED FROM 40 MILLICURIES TO 314 MILLICURIES

Mean 48 Hour Urine Excretion		Correlation Coefficient	Probable Error of C. C.
Tracer	Therapy		
44.1	48.3	0.862	0.033

whether it would be possible to predict from prior tracer studies the response to a large amount of I^{131}. As can be seen from the data already presented, if this prediction is to be accurate there must be good correlation between the integrated blood concentration of I^{131} after therapy and tracer doses and there must furthermore be a good correlation between the amount of radioiodine retained in the body after both therapy and tracer doses. Table II shows the result of a comparison of the amount of I^{131} excreted in the urine after tracer and therapeutic doses given within a few weeks of each other. It can be seen that there is a reasonably good correlation between the amount excreted in forty-eight hours by any one patient after a tracer dose and the amount excreted by the same patient after a therapy dose administered very shortly thereafter. Table III shows the result of a comparison of blood levels of I^{131} obtained in 18 patients given tracer

TABLE III

COMPARISON OF THE INTEGRATED LEVEL OF I^{131} IN THE BLOOD AFTER A TRACER AND AFTER A THERAPEUTIC DOSE IN THE SAME PATIENT

Therapeutic Dose	Millicuries at End of 48 Hour Period*	Tracer rep†	Therapy rep†
52.7 mc.	20.9	20.4	14.0
75.2	36.8	50.5	69.3
100	39.0	136	80.8
151	50.8	76.9	68.2
78.5	52.4	234	228
198	61.0	106	87.3
107	73.7	118	218
107	78.3	178	176
107	85.7	131	158
234	86.6	184	400
201	88.4	106	46.1
182	111	377	371
200	119	61.0	55.0
251	119	16.7	128
200	132	51.1	20.8
264	134	150	255
254	146	33.3	195
247	183	41.0	260

Patients	No. of Cases	Ratio Therapy Blood / Tracer Blood	Correlation Coefficient	Probable Error of C. C.
All	18	1.4	0.650	0.088
Those retaining less than 115 mc. in 48 hours	12	1.1	0.836	0.058

* Millicuries of I^{131} retained by the patient 48 hours after administration of the therapeutic dose.
† Integrated blood concentration of I^{131} assuming both the tracer and the therapeutic dose were 184 millicuries.

and therapy doses within a few weeks of each other. The amount of I^{131} in the blood was followed after both tracer and therapy dose for at least a week. Both tracer and therapy blood rep are for purposes of comparison calculated on the assumption that each dose was 184 millicuries.

It can be seen that there is very poor correlation between the integrated blood concentration of radioiodine in the same patient after a tracer and after a therapy dose administered shortly thereafter. However, if one considers only those doses of which the patient retained in forty-eight hours less than 115 millicuries the ratio between the integrated blood levels is very nearly one and the correlation coefficient is very high. It is desirable to use the amount of I^{131} retained by the patient in forty-eight hours (the amount administered minus the amount excreted in the urine) as the criterion because it is only that I^{131} which is concentrated in thyroid tissue (normal or cancerous) which later gives rise to the circulating protein bound iodine in the blood. We can say, therefore, that in patients retaining less than 115 millicuries of radioiodine in forty-eight hours the amount of I^{131} administered does not affect the integrated blood concentration of the isotope. Since there is also a good correlation in the urinary excretion of radioiodine in patients given tracer and therapeutic amounts it should be possible to predict with some degree of accuracy the amount of damage to the hematopoietic system that will be obtained with any dose of I^{131} of which less than 115 millicuries is retained.

DISCUSSION

Any discussion of these results must have as its prelude a list of a few of the assumptions and sources of error that may be found in this study.

(1) Although the radiation delivered by I^{131} is delivered at a rate of 8 per cent per day on the basis of physical decay, from patient to patient the actual rate of radiation may be quite variable as the concentration in blood and body is constantly changing. Hence, of 2 patients with the same integrated radiation dosage one may have received half the total in four days and the other half the total in about eight days. This variable in rate of radiation dosage has been not only impossible to control but virtually impossible to evaluate statistically.

(2) Calculations of radiation dosage as expressed in this paper are at best rough approximations. A combined rep of 450 (plus a certain body retention factor) may well be in absolute terms perhaps 50 per cent in error (see footnote, page 275). In addition radiation calculated from "body retention" is in error by a factor depending on the geometry of the patient in relationship to the locus where the I^{131} is concentrated. If, for example, 80 per cent of a dose were concentrated in the great toe, the blood would receive a great deal less radiation than if the same amount were concentrated in the chest. All patients studied with one exception concentrated the iodine in some area in the trunk (thyroid, lung, liver, vertebrae, etc.) so this factor is of probably minor importance. The one other patient had a functioning metastasis in the skull. Insofar as the constants used to calculate the rep are the same in each individual study they are most useful for intragroup comparison. Comparison of the rep as herein calculated with roentgens delivered by external roentgen irradiation is unwarranted.

(3) Insofar as there is a rather wide variation in the total lymphocyte count from day to day even 3-point weighted averages will show by the method used a certain maximal fall from pretreatment levels when there has been no radiation and no change in the patient's status. No attempt has been made to evaluate this for the main effect of such a statistical phenomenon would be to shift the final equation on the Y axis without any change in slope.

(4) There was a difference in the patients in their metabolic rate prior to irradiation. Some of the patients were myxedematous, some slightly hypothyroid and some euthy-

roid. Two were hyperthyroid. Blount and Smith[1] have shown that the administration of desiccated thyroid to rats prior to roentgen irradiation decreased the LD_{50}. However Smith and Smith[17] showed that thiouracil or propylthiouracil administered for as long as forty-two days prior to roentgen irradiation had no effect on the mortality rates. The assumption may then be made that hypothyroidism does not affect radiation mortality. Subsequent to the therapeutic dose of I^{131} a few of the patients showed an increase in basal metabolic rate. In none, however, did frank hyperthyroidism supervene. The difference in thyroid status between patients and the change during irradiation may then be assumed to be of minor significance. However, a lymphocytosis in both myxedema and Graves' disease is of frequent occurrence. The role of the change in thyroid status upon lymphocyte production cannot really be evaluated but may be considered a real source of error in this study.

(5) Dougherty and White[2,3] have shown that adrenocortical hormones will depress the lymphocyte count. Furthermore in many of the patients studied the irradiation appeared to be of sufficient magnitude to act as a stressing agent. It has been shown that stress, and specifically that large amounts of whole body irradiation, cause an increased excretion of 17-ketosteroids.[7] Hence a part of the lymphocyte depression measured may be an indirect effect of the irradiation. If this is the case another relatively uncontrolled variable is present.

(6) Another possible source of error is inaccurate estimation of blood I^{131} the first day subsequent ot its administration. In some cases the first value for blood I^{131} has been determined twenty-four hours after the dose. The mean concentration of I^{131} in the blood the first twenty-four hours has been considered to be this value. Obviously within sixty minutes after ingestion the blood I^{131} was much higher and then decreased at a rate proportional to the renal excretion and the thyroid and/or tumor uptake rate. Twelve patients were studied during the first twenty-four hours with frequent blood I^{131} determinations, and the final integrated blood I^{131} concentration obtained in this manner was compared with that obtained by extrapolating the twenty-four hour value to $T=O$. A comparison of this as a source of error in calculation of integrated blood radioiodine concentration in patients excreting various amounts of the isotope is seen in Table IV.

TABLE IV

INTEGRATED BLOOD CONCENTRATION OF I^{131} (EXTRAPOLATED TO INFINITY) IN 12 PATIENTS RECEIVING LARGE DOSES. A COMPARISON OF VALUES OBTAINED BY ASSUMING THE QUANTITY OF I^{131} 24 HOURS AFTER THE DOSE HAD BEEN PRESENT THE ENTIRE FIRST DAY (CALCULATED BLOOD) WITH A VALUE OBTAINED BY ACTUAL DETERMINATION OF THE I^{131} IN THE BLOOD AT FREQUENT INTERVALS THE ENTIRE DAY (OBSERVED BLOOD)

Number of Cases	48 Hour Urine Excretion	Observed Blood / Calculated Blood
3	>70%	1.29
3	<70% >50%	1.13
6	>50%	1.02

In the patients studied for lymphocyte fall all but 2 of those excreting over 70 per cent in the urine in two days had an adequate number of determinations of blood concentration of I^{131} performed in the first twenty-four hours. Hence this factor should not affect the data.

(7) A few of the patients studied received I^{131} in small dosage prior to the dose studied. In all instances prior I^{131} at more than tracer (1 millicurie or less) levels was given more than six months previously. Furthermore analysis of the depression in lymphocytes in these patients showed no consistent variation from that seen in the other patients.

(8) Finally, the number of patients studied is small, ranging in the various comparisons from 18 to 33. The inaccuracy of conclusions based upon small groups needs no amplification.

It is certainly not surprising to discover that there is no simple dose response re-

lationship for I^{131} in humans of varying thyroid states. This would seem to suggest a revision of the plan of administering I^{131} in certain centers where the assumption seems to be made that from a dosage standpoint this isotope can be compared with "aspirin."

The observation that the depression in circulating lymphocytes is a very sensitive indication of radiation has been made many times before. The excellent correlation between radiation dosage and maximal lymphocyte depression has also been noted in experimental animals. McDonald,[11] for example, has investigated the effect of whole body irradiation with slow neutrons on lymphocytes in the rat and found an excellent correlation. Although the data he has published covers only five different dosage levels it is interesting that the points fit the equation $Y=A+B \log X$ quite satisfactorily. Price ,[13,14] on the other hand, investigating the rate of fall in lymphocyte count in patients given roentgen irradiation found that none of the parameters gave any reliable index of the patient's tolerance to radiation. Since any patient's clinical response to irradiation is extremely difficult to quantitate it may be that the lack of correlation between rate of lymphocyte fall and clinical status is due to inaccurate evaluation of the latter variable.

Table V

COMPARISON OF CLINICAL RADIATION SICKNESS (0 TO ++++, SEE TEXT) WITH RADIATION RECEIVED BY THE BODY FROM THE INTEGRATED AMOUNT OF I^{131} RETAINED. THESE THERAPEUTIC DOSES OF I^{131} RANGED FROM 40 MILLICURIES TO 264 MILLICURIES

Body retention in millicurie days	0	+	++	+++	++++
0–100	6	1			
100–200	2	1	3	2	
200–300	1		1		
300–400			1	1	3
400–500					1

Table v embodies an attempt to correlate the "clinical" response to irradiation with radiation dosage. In this chart the following grading of "radiation sickness" has been used:

0—No symptoms or signs
+—Nausea without vomiting
++—Nausea and vomiting lasting one day
+++—Nausea and vomiting lasting more than one day but not requiring intravenous fluid replacement
++++—Nausea and vomiting severe enough to require intravenous replacement of fluids.

It can be seen that only a very approximate correlation is present. In this table the radiation has been calculated only on the basis of I^{131} retained in the body. Even less correlation is seen if the integrated blood concentration of I^{131} is used as a measure of radiation.

The equations which best fit the experimental points deserve some comment. Since three rather different equations fit the points almost equally well it is impossible to use the form of any of these equations as the basis for any theoretical considerations. Since all equations, however, give the best correlation with radiation calculated in the same way (blood radiation plus the 0.09 body retention factor) it may reasonably be inferred that calculation of radiation in this manner is the most accurate of the methods tried.

The marked discrepancy between the integrated concentration of radioiodine in the blood after tracer and therapeutic doses given to the same patient when the therapeutic dose is retained in forty-eight hours to the extent of more than 115 millicuries is of some interest. In these cases it appears that radiation has had a direct effect on the metabolism of iodine. Studies by Robbins *et al.*[16] in several of these patients have shown that as much as 88 per cent of the blood radioiodine after these very large doses is not thyroxine. After tracer and smaller therapeutic doses of I^{131} almost all the blood radioactivity appears

to be thyroxine. Additional chemical studies by Robbins have shown that the abnormal product is probably thyroglobulin that has leaked from the badly damaged thyroid follicle. As this only occurs with severe radiation damage the discrepancy between tracer and therapy blood concentrations will be seen only with very large doses of I^{131}. Obviously poor retention of any dose, no matter how large, would not be accompanied by thyroglobulin release. Similarly we might expect to see thyroglobulin release with relatively small doses of I^{131} if there were excellent retention of the isotope in a small amount of tissue.

SUMMARY

1. Thirty-three patients have been studied with tracer and very large therapeutic doses of I^{131}.

2. The lymphocyte count has been found to be the most sensitive and reliable index of the degree of radiation damage.

3. The lymphocyte fall after large doses of I^{131} is not well correlated with the millicuries of the isotope administered but is very well (correlation coefficient 0.919) correlated with the integrated blood concentration of this isotope plus an empirical factor derived from the integrated amount of isotope retained in the body.

4. In patients retaining in forty-eight hours less than 115 millicuries of radioiodine it has been possible to predict from tracer studies the amount of radiation that a large dose of I^{131} will deliver.

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A COMPARISON OF THE REACTIONS OF THE IRRADIATED PARTS OF THE BODIES OF TWO DAY OLD MICE AND URODELE AMPHIBIANS TO ROENTGEN TREATMENT*

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TO UNDERSTAND the biological effects of roentgen rays, it is important to study not only a particular type of reaction in one organism, but to compare the effects of irradiation upon a number of different organisms to attempt to perceive some basic principle underlying the response of animals to irradiation in general.

The purpose of this work is to study the response of the two day old mouse limb to roentgen irradiation and to make a comparison with the reactions of the amphibian limb. It was important to carry out the irradiations in both animals under the same conditions. The conditions for the irradiation of amphibian limbs were as follows: 43 kv. max., 3 ma., without filter, and (in more recent work) 53 kv. max., 5 ma., without filter. A comparison of the effects of irradiation under both conditions has shown that these conditions, as far as the response is concerned, may be considered to be identical. The conditions for the irradiation of mouse limbs were the same as in our more recent work with amphibians (53 kv. max., 5 ma., without filter).

The animals were fixed by means of a saturated solution of bichloride of mercury with 5 per cent acetic acid. The fixed material was embedded in paraffin, and sections were made at 10 μ. The sections were stained with Ehrlich's hematoxylin and eosin.

Definite signs of reaction to irradiation were noted in mice as early as the seventh or eighth day after treatment with 3,000 roentgens. By this time, the growth of the hair in the treated region was completely suppressed, and retardation of growth of the treated limb was already noticeable.

Histological examination of the treated limbs of animals in these experiments showed that the most pronounced damage always occurred in the cartilaginous epiphysial plates (zone of transformation of cartilage to bone). The first alteration in this region of a limb was noted eight days after irradiation with 3,000 r (Fig. 1*C*). The zone of proliferation of cartilage cells was only partially damaged. In this region, the columnar arrangement of cells was disturbed. But in the adjacent zone of cartilage calcification, the greatest alteration had already started at this time (Fig. 1*C*). Although bone formation would normally be in progress here, none was observed. This zone was much larger than usual and consisted of very large cells. This appears to be the first step in a process of degeneration resulting from roentgen-ray damage which progresses gradually and leads to the formation of giant cells. The formation of such giant cells is typical and is a widely distributed phenomenon; one which has been described in various irradiated organisms by a number of authors (Cramer,[19] Ludford,[36] Scheremetjewa and Brunst,[44] Helwig,[28] Pietschmann,[41] Brunst,[5] Dederer,[20] Luther,[37] Lasnitzki,[33] Hall and Friedman,[27] Brunst,[6] and others).

Much more definite damage of the limb was observed twenty-four days after irradiation with 2,000 r (Fig. 2*A*). The damage was more clearly seen in the zone of proliferation of the epiphysial plates, where there was a complete disorientation of

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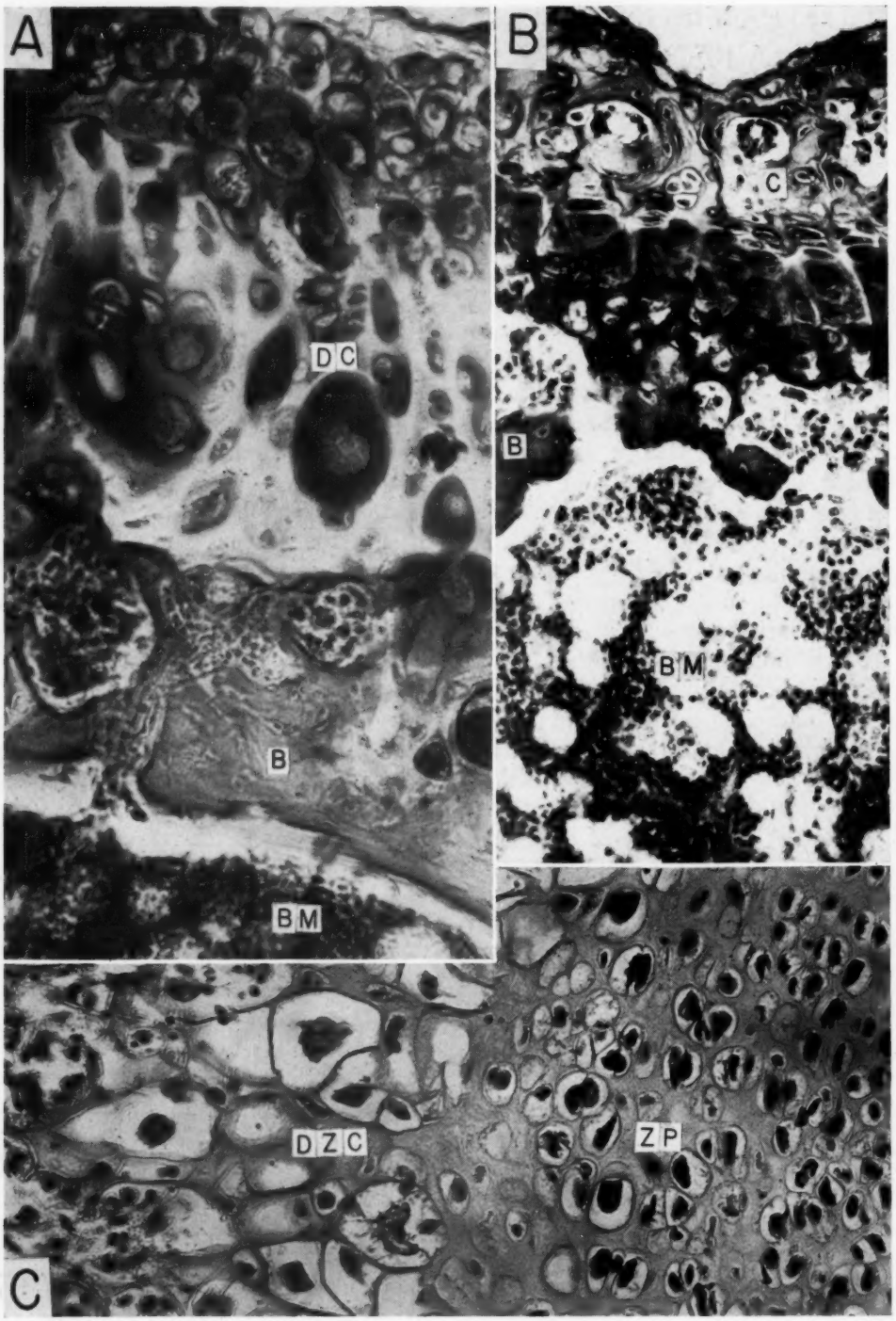

FIG. 1. (*A*) Frontal section through the tail vertebrae of an animal ninety-eight days after irradiation with 3,000 r. B—bone; BM—bone marrow; DC—degenerating cartilaginous tissue.

(*B*) Frontal section through the tail vertebrae of control animal from the same litter as the treated animal in (*A*). B—bone; BM—bone marrow; C—growing cartilaginous tissue.

(*C*) Frontal section through the hind limb of an animal eight days after irradiation with 3,000 r. DZC—beginning of formation of giant cartilage cells in degenerating zone of calcification; ZP—zone of proliferation of cartilage. Note atypical appearance. Photomicrographs ×280.

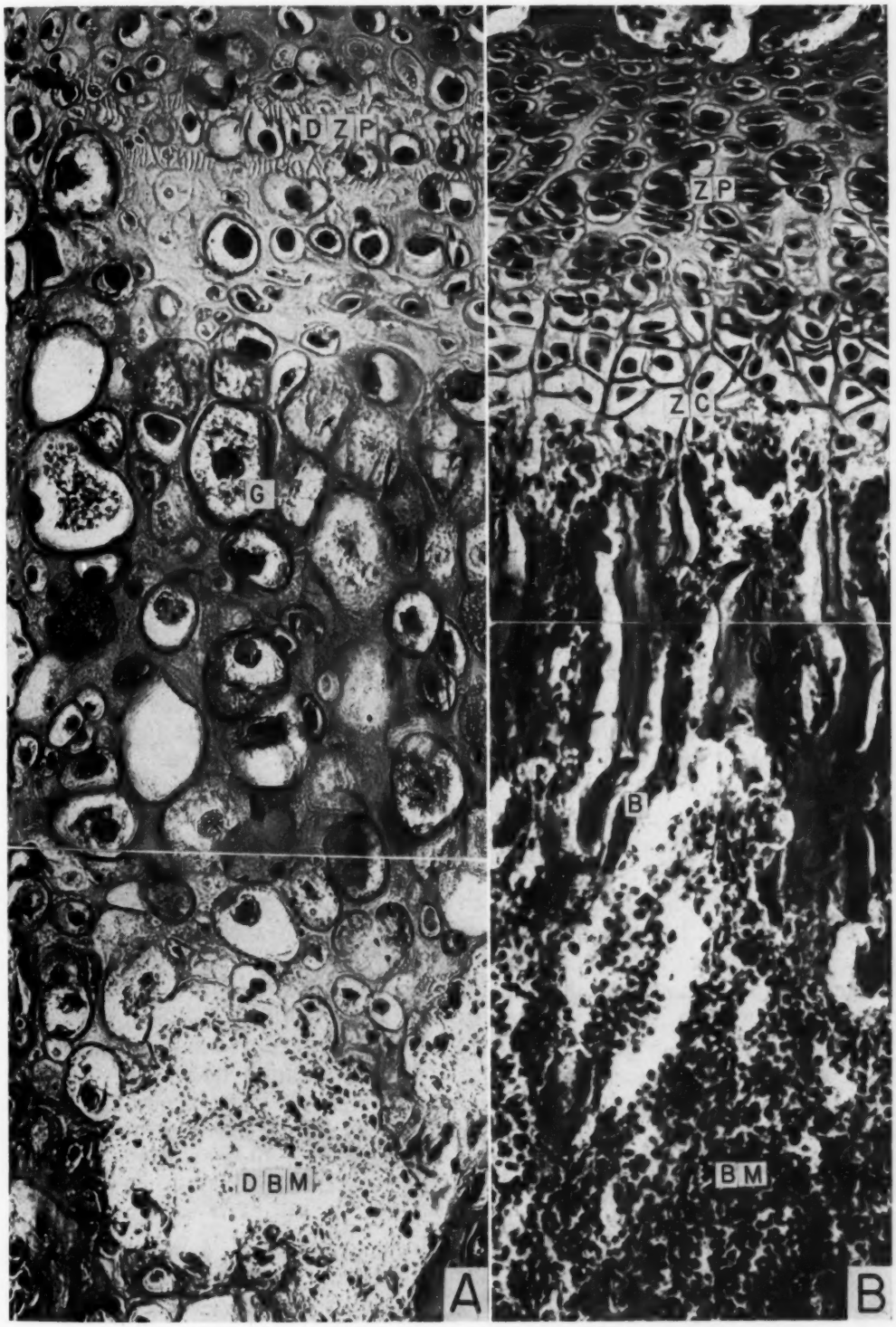

Fig. 2. (*A*) Frontal section through the hind limb of an animal twenty-four days after irradiation with 2,000 r. DBM—destroyed bone marrow; DZP—damaged zone of proliferation of cartilage; G—giant cartilage cells obviously developed from the zone of calcification.

(*B*) Frontal section through the hind limb of control animal. B—bone trabecula; BM—bone marrow; ZC—zone of calcification of cartilage; ZP—zone of proliferation of cartilage. Photomicrographs ×280.

cartilage cells. More active alterations were observed in the zone of calcification of the cartilage. If one compares the histological pictures at eight and twenty-four days after treatment (Fig. 1*C* and 2*A*), it is seen that at twenty-four days after irradiation, this zone is almost twice as large as it is at eight days, and the cells are much larger (Fig. 2*A*). These are true giant cells. In many cells, the nucleus is broken into several pieces or may be completely fragmented. It is obvious that the nucleus has gradually disintegrated in some cells. The process of endochondral ossification has completely stopped. The general picture of the irradiated epiphysial plate contrasts sharply with the normal histological picture of a control animal of the same age (Fig. 2, *A* and *B*). The vertebrae in the treated tail are much shorter than in the control animal. This is the result of growth suppression of the skeleton. Examination at high magnification shows a clear difference between irradiated and nonirradiated vertebrae (Fig. 1, *A* and *B*). The cartilaginous region in the treated vertebrae is very large and consists of giant cells. It is clear that the processes of growth and endochondral ossification have been completely arrested. In the control animal, however, the cartilaginous region is relatively small, and it is seen that ossification and growth are normal (Fig. 1*B*). In the central region of the skeletal tissue of some irradiated limbs between bone and cartilage, there was found a comparatively large cavity containing degenerated cells. Such cavities in other animals finally became very large and were obviously the result of complete degeneration and death of numbers of cells. Retardation of development as a result of local roentgen irradiation in mammals was observed also by other authors (Krukenberg,[32] Försterling,[22,23,24] Walter,[48] Bagg and Halter,[1] Brooks and Hillstrom,[4] Hinkel,[29] Reidy, Lingley, Gall and Barr,[43] and others). Försterling[22] investigated the effect of local roentgen irradiation upon the development of the limb of an eight to nine day old dog. Seven and one-half months after treatment, the difference in length between irradiated and nonirradiated limbs was 8 cm. In another paper, the same author[23] described the retardation of development of an irradiated limb and part of the head of a goat. He observed an inhibition of development in the treated region of the head, including the eye (which was only one-third to one-fourth of the normal size) and horn. Regen and Wilkins[42] investigated the effects of irradiation with 2,600 r upon the development of left forelegs of twenty-five day old rabbits. Roentgenograms were taken at intervals of one month. It was found that bone growth could be completely stopped by treatment with such a dosage of roentgen rays. Some authors have emphasized that the epiphysial plate is a very radiosensitive tissue (Barr, Lingley and Gall[2]). Others have noted in irradiated parts a definite enlargement of the cartilaginous portions, which consist of large cystiform cells (Baunach[3]) and have described the disorientation of the columnar arrangement with an arrest of growth of cartilage (Gall, Lingley and Hilcken[25]). Gates[26] noted an early alteration of cartilage cells and an inhibiton of endochondral ossification following irradiation. He described the earliest changes in epiphysial cartilage cells, such as swelling and loss of columnar pattern. Hinkel[29] described the effect of irradiation with 600 r on thirty day old albino rats. One week after treatment, the cartilage cells were abnormally large and bizarre and poorly columnated. Two months after treatment, the columnation had been restored. It is clear that a dosage of 600 r was too small to show a great effect in thirty day old animals. It appears to us that the formation of large cells, swelling of cells, and formation of cystiform cells in treated cartilage, as described in mammals by the authors mentioned above, are the same phenomena which are termed the formation of degenerating giant cells in the present paper.

A comparison of this phase of development of the tissue reactions to irradiation

in mammals with those in urodele amphibians leads to the following conclusions:

1. The reaction to roentgen irradiation is to be noted much earlier in mice than in amphibians (seven to eight days in mice and eighteen to twenty days in amphibians; see Brunst,[5,6,7,8] and Brunst, Barnett and Figge[9]). It is possible that this is a result of the more active metabolism in warm-blooded animals.

2. The process of reduction (a more or less rapid decrease in size of the treated parts) which was observed nearly always after the irradiation of young regenerating or developing limbs in urodele amphibians (Brunst[5,6,10]), was not observed in mice. In amphibians, the reduction process is typical for the irradiated, rapidly growing, young parts, which consist of relatively young, undifferentiated cells. But it may be that this process is one particular case of the "primary acute reaction" which is more typical for amphibians. Certain amphibians (anuran tadpoles) characteristically show the reduction process at a definite stage of normal development; namely, in the reduction of the tail. It is noteworthy that the reduction process in urodele limbs was observed not only after roentgen irradiation, but also under other conditions (for example, after the destruction of the innervation of the limbs. See Schotté,[45] Brunst,[11] Butler and Schotté,[13] Schotté and Butler,[46] and others).

3. One may compare the alterations which were observed in the tissues of treated mice during the thirty to forty days following irradiation with the "primary acute reaction" of amphibians. In the latter animals, this reaction began about ten days later than in mice but continued for approximately the same length of time, *i.e.*, about thirty to thirty-five days (see Brunst[6,7,8]). It is noteworthy that some phenomena which are observed during this period in the mouse are temporary and are typical only for this "primary reaction." These are: a temporary suppression of hair growth and a destruction of bone marrow which is completely regenerated later. In the mice, we did not observe an inflammatory reaction or an invasion of macrophages in the treated region; reactions which are quite typical of the "primary acute reaction" in amphibians (see Brunst[5,6,7,8] and Brunst, Barnett and Figge[9]). The reason for our failure to observe these reactions in mice if they occur cannot be clarified at present, but this inflammatory reaction, after roentgen irradiation, has been observed in mammals by other authors (Ewing,[21] Cramer,[19] and others). It will be necessary to reinvestigate this problem.

Although there may be some doubt concerning the comparison of the primary alterations after roentgen irradiation in the mouse with the "primary acute reaction" in amphibians, we can make a clear comparison of the subsequent alterations which occur. In mice fixed ninety-eight days after treatment, there is observed a partial recovery of the tissues. Bone marrow has completely regenerated and is normal in appearance. At this time, hair growth has almost returned to normal, but the coat is gray in color. The graying of the hair in mammals was observed by many authors after roentgen irradiation (Keeler,[30] Cameron,[17] Lübnow,[35] Chase,[14] Chase, Quastler and Skaggs,[15] Chase,[16] and others). It was noted that some tissues (epithelial, areolar, and perhaps muscular) were almost always normal in appearance. In the zone of transformation of cartilage to bone, however, it was possible to see clearly the damaging effects of irradiation. This region consisted of large degenerated cells. There was histological evidence that there was no growth of the bones, which agreed with the observation that the irradiated parts failed to grow.

It is clear that in the treated parts of the mouse, one can observe, at this time (ninety-eight days after treatment), the same state which is found in treated amphibians sixty days after irradiation and thereafter. This state has been termed the "stable state" (Brunst[6,7,8] and Brunst, Barnett and Figge[9]). In this state, it is typical to find treated parts with a normal appearance, both grossly and microscopically, but exhibiting a complete arrest of mitotic activity and development. There is one notable difference between the reactions of mice and amphibians; namely, in mice some tissues show a distinct evidence of roentgen cell damage during the "stable state," while in amphibians they do not. It is quite possible that the cells of amphibian

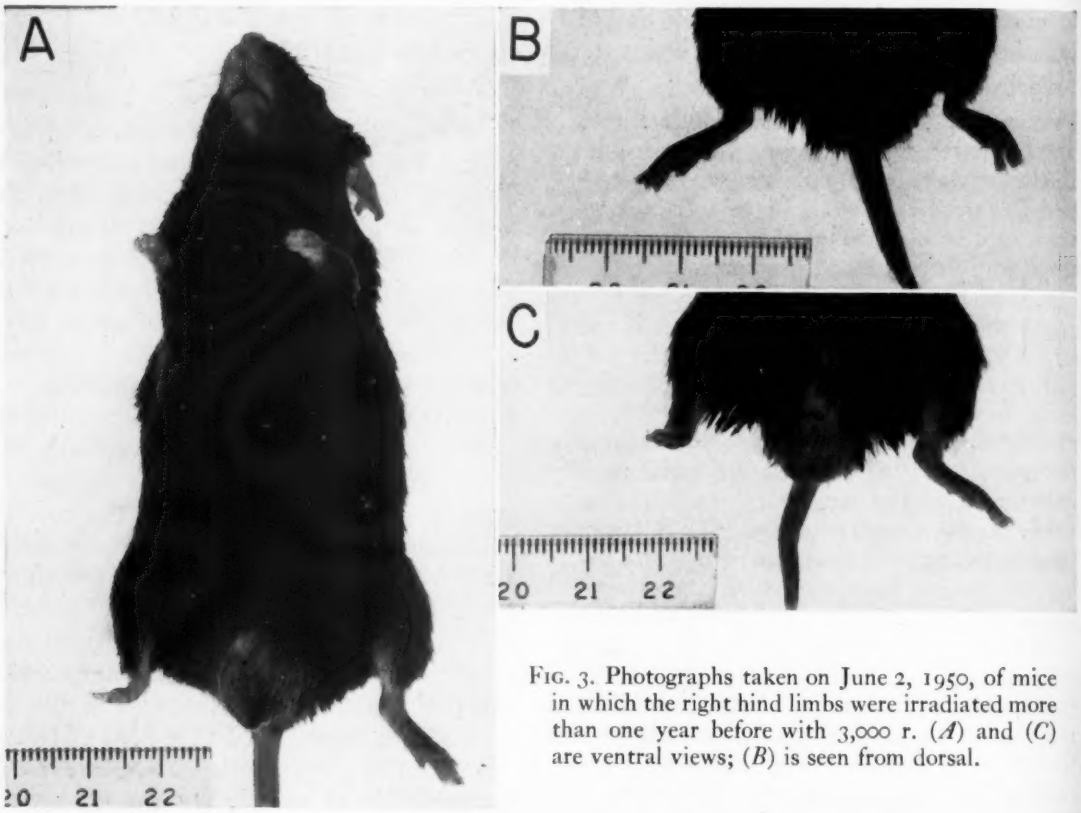

FIG. 3. Photographs taken on June 2, 1950, of mice in which the right hind limbs were irradiated more than one year before with 3,000 r. (*A*) and (*C*) are ventral views; (*B*) is seen from dorsal.

tissues are better able to recover after roentgen irradiation in keeping with their well known ability to regenerate. Roentgen rays definitely suppress the ability of limbs to regenerate in amphibians (Brunst[5,6,10]), but cellular regeneration or repair is probably only partially inhibited. It may be that some cells, in spite of great damage, can recover completely. Other cells with still greater damage disappear completely because of the phagocytic activity of the macrophages (Brunst[5,6,10]). It is possible that phagocytosis is much more active in amphibian tissues than in those of the mouse. A peculiarity of this sort in amphibian tissues could perhaps explain this difference in the appearance of treated amphibian and mouse tissues.

It was shown in previous papers (Brunst[5,6,10]) that irradiated amphibian limbs retain their viability for long periods of time (more than one year), while maintaining the normal appearance of the treated parts but with a suppression of regeneration. It is noteworthy that if treated tissues remain alive for a very long time, in some cases a very late radiation reaction may arise. Such a case was described by Matas,[40] who observed a lethal gangrenous dermatitis in a man four years after irradiation. In amphibians, however, no late reaction to irradiation could be observed, and limbs which were treated with 7,000 r retained their normal appearance for several years (Brunst[5,6,10]). Some authors think that although it is possible to observe long continued suppression of growth of treated parts without impairment of viability in amphibia, it does not follow, however, that the same is to be observed in the mouse. Luther,[37,38,39] from his observations on the effects of roentgen rays on tumor development in mammals, concludes that this phenomenon cannot be observed in these mice (Luther—personal communication). However, our own observations have shown that this phenomenon may be seen in mice as well as in amphibians.

The right hind limbs of several two day old mice were irradiated with 3,000 r. More than one year later, these mice were photographed, and the treated parts were fixed. The photographs demonstrate that the treated limbs have a normal appearance (Fig. 3, *A*, *B*, *C*) except that in most cases, the growth was completely arrested (Fig. 3, *A* and *C*). Only in certain cases was the growth partially inhibited (Fig. 3*B*). However, our experience shows that such cases are the result of improper placement of the animals during irradiation so that the dose of roentgen rays was not sufficiently large to produce a complete suppression of limb growth.

Histological examination of control limbs (untreated hind limbs of animals irradiated on one side only) showed a completely normal structure. All tissues have an appearance which was typical for normal animals (Fig. 4). An investigation of irradiated limbs with arrested growth showed that some tissues were almost normal in appearance, but others showed great degenerative alterations. The skin was normal in appearance, but the hair was almost completely undeveloped (Fig. 5, *A* and *B*), while in the corresponding portion of an untreated limb, the hair was well developed (Fig. 4*B*). The areolar and adipose tissues were essentially normal in appearance. Even the cartilage in some places appeared to be normal (Fig. 5*A*, NC). But the greatest disturbance was observed in certain adjacent regions of cartilage and bone. It is noteworthy that the same process of degeneration, which was described in the epiphysial plate region of cartilage (zone of transformation of cartilage to bone) in

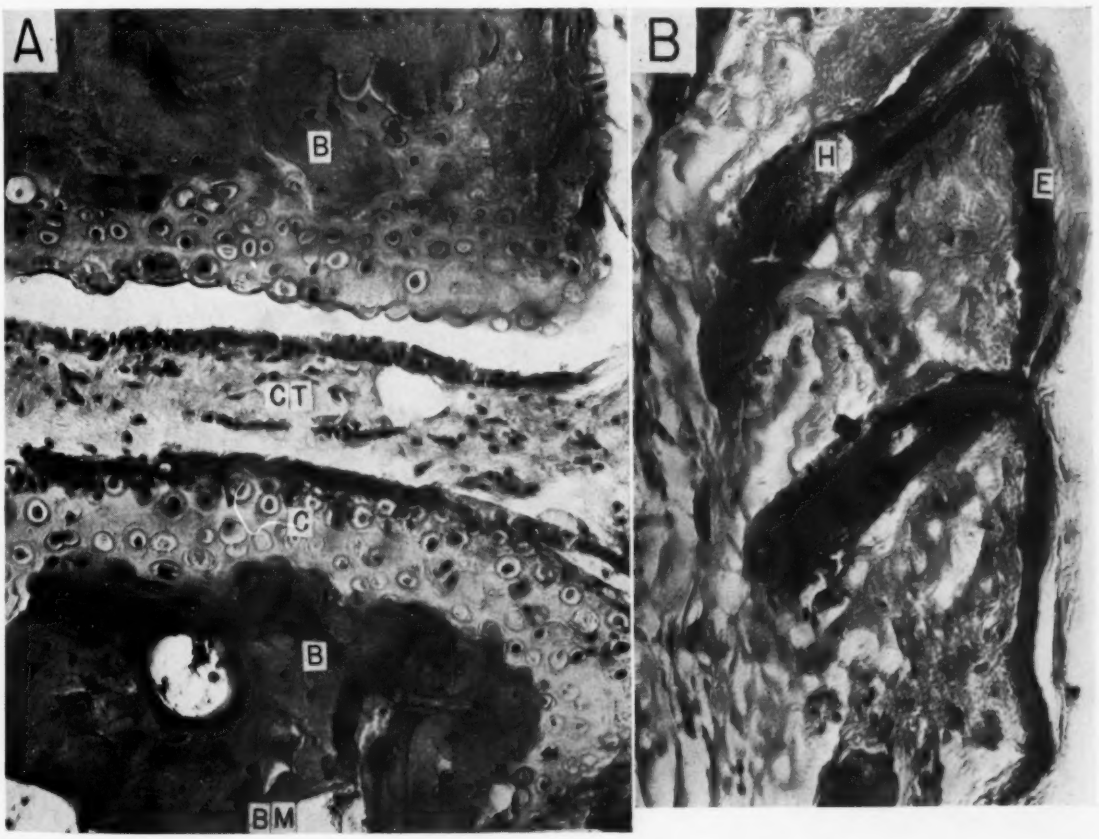

Fig. 4. (*A*) (*B*) Longitudinal sections through the control limb of a mouse. The fixation was made simultaneously with the irradiated limb. B—bone; BM—bone marrow; C—cartilage; CT—connective tissue (areolar tissue); E—epithelium of the skin; H—hair. Photomicrographs ×280.

Fig. 5

mice ninety-eight days after treatment, progressed very slowly for more than one year. A number of the treated cells apparently degenerated completely and died, leaving large cavities containing several degenerated cells (Fig. 5, *A*, *C*, *D*). These dead cells were probably removed by means of lysis or phagocytosis. We have not observed either of these phenomena in the mouse, but phagocytosis after roentgen irradiation has been observed in insects (Carothers[18]), amphibians (Litschko,[34] Brunst[5,6,12]), and mammals (Tsuzuki,[47] Kosaka[31]). According to Withers,[49] the cells killed by irradiation are removed by autolysis and phagocytosis. A very large cavity of this type was found in a limb which was fixed one year and twenty-two days after irradiation with 3,000 r (Fig. 3*C*). A contour drawing of the limb, made with the aid of a camera lucida, illustrates the size of these cavities (Fig. 6). It is necessary to emphasize, however, that some cartilage cells, in spite of great abnormality and damage, were nevertheless viable and lived for a long period of time (more than one year). Most of these cells are abnormally large giant cells and therefore, are easily distinguished from other cells (Fig. 5, *A*, *C*, *D*).

The present investigation shows that, in spite of the great difference between the mouse and urodele amphibians, a great similarity in the biological effects of roentgen rays in the two forms was observed. In both cases, following a latent period, temporary and comparatively acute alterations in the tissues ("primary acute reaction") were observed together with a retardation or complete suppression of mitotic activity and growth. About fifty days after the treatment in both cases, there was a more or less complete recovery of the tissues. In

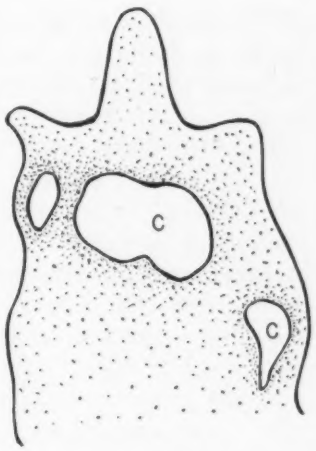

Fig. 6. Contour of the limb with suppressed growth one year and twenty-two days after irradiation with 3,000 r. C—cavity which was observed at this time in treated tissues. Semischematic drawing made with the aid of a camera lucida.

amphibian tissues, this process of recovery was much more active, and therefore all the treated tissues appeared normal ("invisible injury"). In the mouse, however, some of the tissues appeared normal, but in others, a continuing process of degeneration was clearly seen ("visible injury"). But in both animals, in spite of the complete suppression of mitotic activity and development, it was observed that the treated tissues were viable for a long period of time. This "stable state" is similar in both animals.

The most important biological effect of roentgen rays is a suppression of mitotic activity and therefore a suppression of growth and development of the treated parts.

SUMMARY

The purpose of this work was to study the reactions of the two day old mouse limb to roentgen irradiation and to make a comparison of these reactions with those of the urodele amphibian limb. The irradia-

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Fig. 5. Longitudinal sections through a treated limb more than one year after irradiation with 3,000 r. The animal is the one shown in Figure 3*C*. (*A*) General appearance of the tissues in the central portion of the tarsus with a part of the skin. (*B*) Part of the skin epithelium. (*C*) and (*D*) Tissues showing degenerated cartilaginous cells. C—cavity which appears possibly as a result of the complete destruction of parts of the tissues; CT—connective tissue (areolar tissue); E—skin epithelium; G—giant degenerating cartilaginous cells; NC—cartilage which is almost normal in appearance. Photomicrographs ×280.

tions in both animals were made under the same conditions. The right hind limbs of two day old mice were irradiated with 3,000 roentgens. More than a year after treatment, the limbs have had a normal appearance, in spite of a complete arrest of growth. Histological examination of such limbs showed that some tissues were almost normal in appearance, but in others there were observed great degenerative alterations. Most of these degenerating cells were of abnormally great (giant) size and, in spite of this abnormality, were viable and survived for a long period of time (more than one year).

This investigation showed that, despite great differences between mice and urodele amphibians, the effects of roentgen rays upon the two forms were similar. In both animals, temporary and comparatively acute alterations ("primary acute reaction") were observed following a latent period. Subsequently, a more or less complete recovery of tissues occurred in both animals. In amphibian tissues, as a result of this process, all the tissues had a normal appearance ("invisible injury"). In the mouse, some tissues showed distinct evidence of degeneration ("visible injury"). But in both animals, these latter conditions may be called a "stable state," in which it is typical to find a complete suppression of mitotic activity and therefore a complete suppression of growth and development of the treated parts for a long period of time.

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CALORIMETRIC EVALUATION OF THE ROENTGEN FOR 400 KV. AND 22.5 MEV. ROENTGEN RAYS[1,2,3]

By J. S. LAUGHLIN,[4] J. W. BEATTIE, W. J. HENDERSON *and* R. A. HARVEY

ROENTGEN rays were first detected through their action on fluorescent material, and since then their action on other detectors such as photographic film, pastilles, resistance of irradiated selenium, and in the production of ionization has been quantitatively investigated.[23] Of these, ionization measurements have been primarily employed for quantitative determinations of both radiation intensity and dose. When roentgen rays are absorbed in any medium, their energy is imparted to secondary electrons. These electrons lose their energy through ionization and excitation of the atoms of the absorbing medium. Unless chemical change results, all of this energy is eventually converted into heat energy. The average energy lost by an electron per ion pair formed has been determined accurately for various substances and for a medium such as air is fairly constant over a wide range of energy of the electron. Consequently a measure of the ionization can be related to the energy absorbed from the beam (energy dose), and to the energy flux (intensity) of the incident beam. Moreover, ionization measurements in air are reproducible and the techniques well developed.

Therefore, in the absence of a well established technique for measuring the energy output directly, the choice of an ionization measurement as the fundamental unit for the calibration of radiation intensity and dose was natural. The definition of "the roentgen" requires the collection of the ions formed by the secondary electrons which originate in a unit volume due to the absorption of roentgen rays passing through the unit volume. Although the range of a 50,000 volt electron in air is only about 4.5 cm., the stopping power for higher energies is less and a 500,000 volt electron, for example, has a range of about 160 cm., considerably more than 10 times longer. The difficulty in collecting all ions produced by the absorption of roentgen rays above 400 kv. in an ion chamber of reasonable dimensions is apparent. As the energy of the radiation is increased, the measurement of the true roentgen becomes more difficult and its interpretation more ambiguous. These limitations of the roentgen have long been recognized.[4,19,22,24]

It is important to distinguish between intensity and dose. Intensity refers to the flux in the beam and thus applies to the output of generators. It can be expressed in ergs/cm^2-second or in watts/cm^2. Dose refers to the energy absorbed in any particular region of the tissue and can be expressed in ergs/gram or in joules/gram. It is unfortunate that the use of the roentgen for both intensity and dose measurements has tended to prevent a clear distinction between these separate physical entities. The use of separate units for intensity and dose will help distinguish and clarify these distinct physical quantities.

In order to provide a means of directly determining the energy output of roentgen-ray generators which would be free of the limitations mentioned above, a calorimetric method of measurement has been developed. This calorimetric method has proved to be extremely sensitive and accurate, and capable of easily reproducible measurements. It is sufficiently sensitive to be applicable to any roentgen-ray generator in use, and the apparatus is also portable.

Historically, application of calorimetric

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[2] Presented at the Fifty-second Annual Meeting, American Roentgen Ray Society, Washington, D. C., Sept. 25–28, 1951.

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methods has rarely been attempted for roentgen-ray detection. The output of the 50 kv. beryllium window generators is so high that ion recombination made ionization measurements unreliable. A precision calorimetric measurement was therefore made for such a generator by Ham and Trout.[8] Earlier, Stahel[26] had used a conventional calorimeter to measure the absorption of gamma rays from a radium source in lead. The first calorimetric determination of high energy roentgen-ray flux was made by Kerst and Price,[10] who used thermocouples to measure the flux of the 300 mev. betatron beam. Their success encouraged us in the execution of our plans.

In 1948, a set of pairs of carbon, aluminum, copper, and lead absorbing cylinders with embedded calibration heating coils was built for a calorimetric determination of the energy in the external electron beam in Urbana, Illinois. These measurements have been delayed by attention to other problems related to the clinical application of the betatron. Our first attempt was made with the lead cylinders using thermistors to detect their change of temperature. An audio-frequency and a pulsed bridge arrangement was used with an A.C. amplifier to amplify the bridge unbalance which appeared on an oscilloscope. Though the heating effect was detectable, sufficient "noise" penetrated our shielding to seriously limit the accuracy. We then successfully employed a D.C. bridge with a high sensitivity galvanometer as a null detector for our first measurement of the energy flux in the betatron beam. The present version of our apparatus employs a special amplifier rather than a galvanometer to amplify the bridge unbalance. The details of our apparatus and results will be described in this report.

THERMISTORS

The temperature sensitive elements employed are thermistors, or thermally sensitive resistors. Their development and properties have been described[1,3] by researchers with the Bell Telephone Laboratories and the Western Electric Company. Thermistors are made of metallic oxides which are semi-conductors. Extensive research has developed them with stable characteristics and free from the many defects such as polarization, which had earlier made them unreliable. Our thermistors are made from a mixture of the oxides of manganese, nickel, and cobalt. They are shaped as rods approximately 4 cm. long and 3 mm. in diameter with electrodes attached at each end. They have a high negative temperature coefficient of resistivity of about -3.9 per cent/degree C.

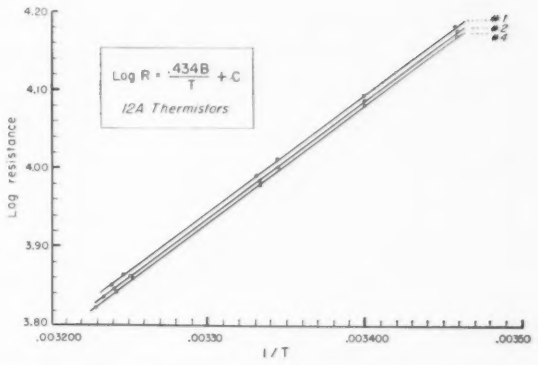

FIG. 1. Plot of logarithm of resistance of type 12A thermistors versus reciprocal of the absolute temperature. Plot of thermistor #3 falls between that of #2 and #4.

Figure 1 is a plot of our measurements of the logarithm of the resistance of our 12A thermistors as a function of temperature. The linear plots can be represented by the expression:

$$R = R_0 e^{B(1/T - 1/T_0)} \qquad (1)$$

where R is the thermistor resistance in ohms at any Kelvin temperature T, R_0 is the resistance at temperature T_0, B is a constant, and $e = 2.7183$. The value of B for all four thermistors tested was $B = 3.458 \times 10^3$ degrees. Figure 2 contains a plot of the resistance on a linear scale versus temperature which exhibits the magnitude of the negative coefficient. Since in each complete experimental "run" the total resistance change was never more than 3 ohms and the temperature change was less than 0.01°C., the resistance-temperature

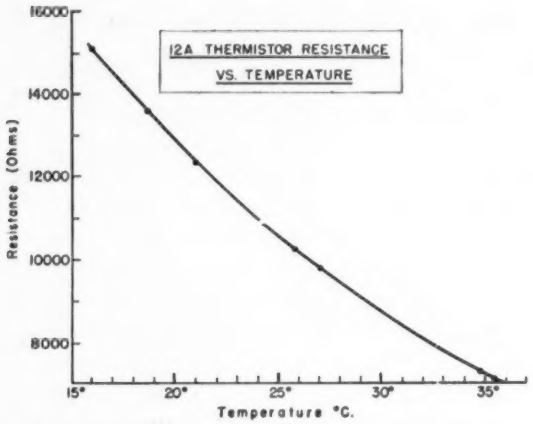

FIG. 2. Plot of resistance of a type 12A thermistor versus temperature. Range employed in each experimental run was about 2.5 ohms in resistance and about 0.0065 degree in temperature.

relation during each run was essentially linear. In our experience the thermistor characteristics have been reproducible and constant.

CALORIMETER

The calorimeter was designed with the objective of absorbing the incident radiation in a body which was thermally insulated from its environment. The temperature change in the absorbing body is measured by means of the resistance change in an embedded thermistor.

For the 400 kv. roentgen-ray experiment the calorimeter cylinder absorbs the total incident beam. Its construction is shown in Figure 3. It consists of a silver-plated brass shell filled with lead. The brass container is made in two sections, and each filled with lead. The outside diameter of each section is 6.2 cm. while one is 5 mm. thick and the other is 2.90 cm. thick. A spiral groove is cut on the lead face of the latter section. A constantan wire heating coil is inserted in this groove with copper leads leading to the exterior. Low melting point bismuth solder is used to join the two sections. The depth of the heating coil from the front surface is approximately the same at which the local ionization is 50 per cent of the surface ionization.

Since the diameter of the incident collimated 400 kv. (peak) beam was 3.0 cm., no radiation could escape through the sides or rear of the cylinder. To examine this point, a lead phantom consisting of 1 mm. lead sheets with film inserted between the sheets was exposed to the beam. Densitom-

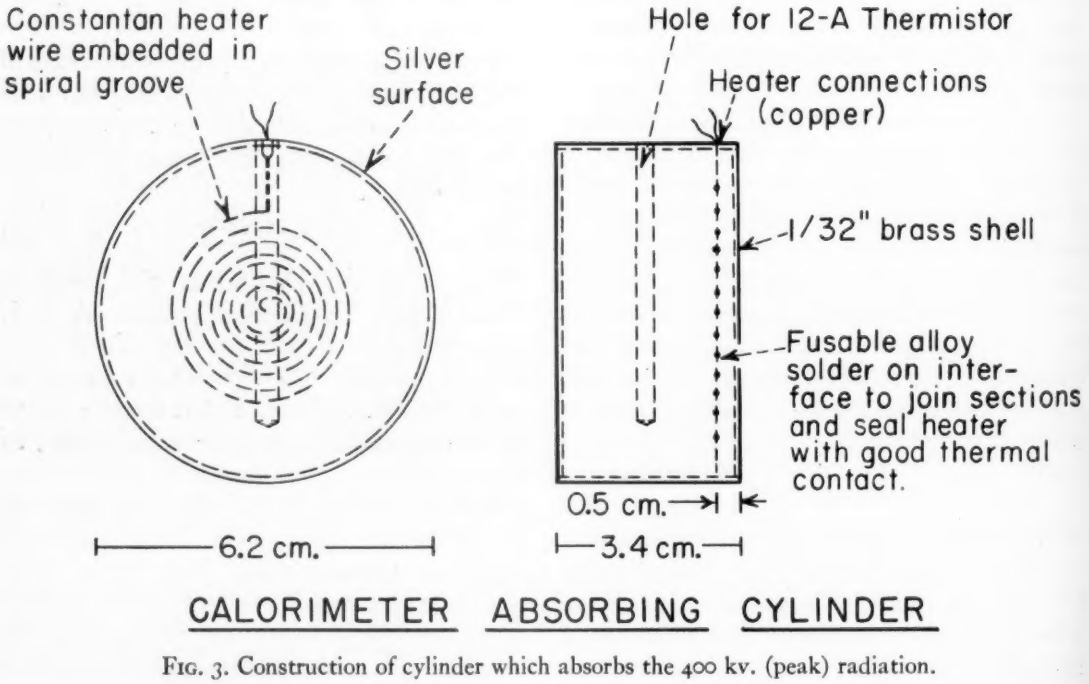
Constantan heater wire embedded in spiral groove
Silver surface
6.2 cm.
Hole for 12-A Thermistor
Heater connections (copper)
1/32" brass shell
Fusable alloy solder on interface to join sections and seal heater with good thermal contact.
0.5 cm.
3.4 cm.
CALORIMETER ABSORBING CYLINDER

FIG. 3. Construction of cylinder which absorbs the 400 kv. (peak) radiation.

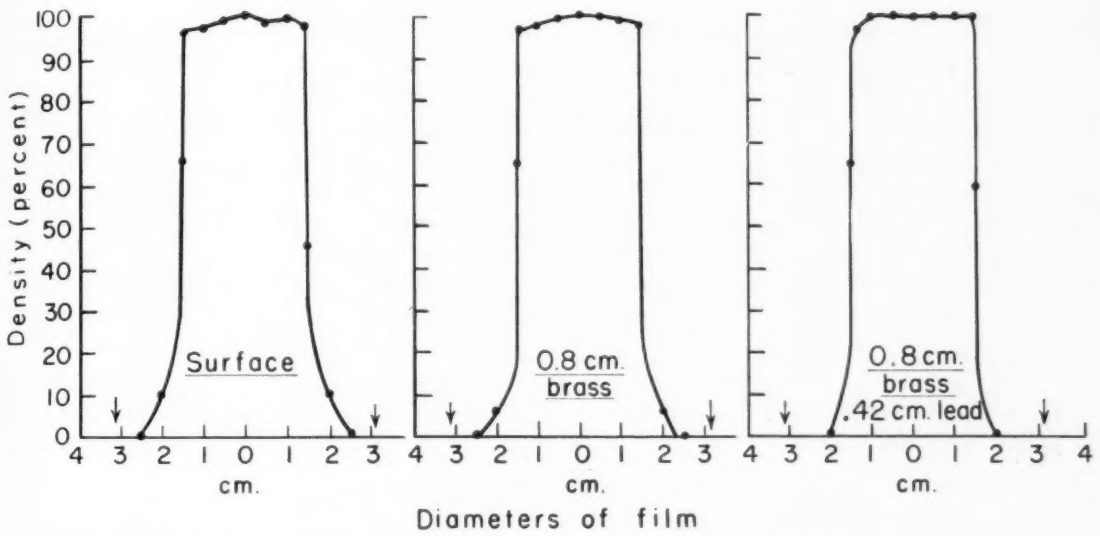

Fig. 4. Densitometer measurements of films inserted between lead sheets of a phantom exposed to the collimated roentgen-ray beam. Depth in phantom is indicated. Arrows indicate edge of absorbing cylinder.

eter measurements on these films indicated that no radiation approached the walls of the cylinder. Figure 4 displays densitometer plots at various depths in the cylinder which indicated a negligible amount of energy was lost through the walls. The measurement of backscattered radiation will be described later.

In use, each cylinder is suspended with its axis along the axis of the roentgen-ray beam. The thermistor is tightly waxed in a vertical hole in the cylinder 1 cm. back of the heating coil. The suspended cylinders are shown with their connections in Figure 5.

Two such identical cylinders are suspended inside a brass silver-plated box which is diagrammed in Figure 6 and shown in Figure 5. This box contains a partition to prevent transfer of radiation between the cylinders. Two windows are located on the box so that either cylinder can be irradiated with an external beam. The window material is thin (.065 gram/cm^2) transparent plastic (plastacele) which is vacuum tight. Thin aluminum foils are placed over the transparent windows. The cylinders are so suspended from three points on each one that they cannot swing or change their position.

All connections and suspensions are attached to the cover or lid of the box. The electrical connections to the thermistors and heating coils are brought through the lid by means of Kovar seals. The vacuum seal between the lid and box is made with a gasket of solder, which is impervious to oil. The vacuum connection is also made to the lid. The whole calorimeter box is

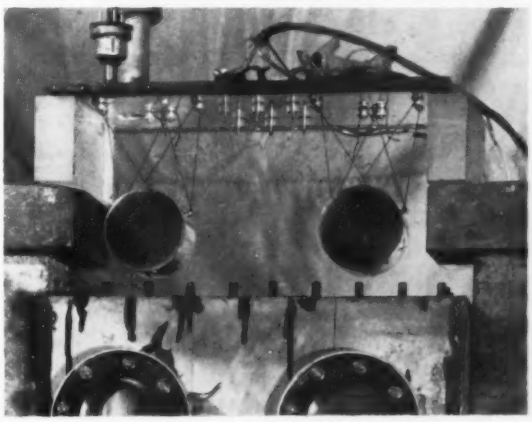

Fig. 5. Photograph of calorimeter disassembled. The suspension of the cylinders from the lid of the calorimeter is shown. Electrical connections pass through the Kovar seals in the lid. Cadmium solder is used on all junctions to minimize thermal electromotive forces. A lead gasket is used for the seal between the lid and container. Calorimeter fits into masonite box which is filled with oil.

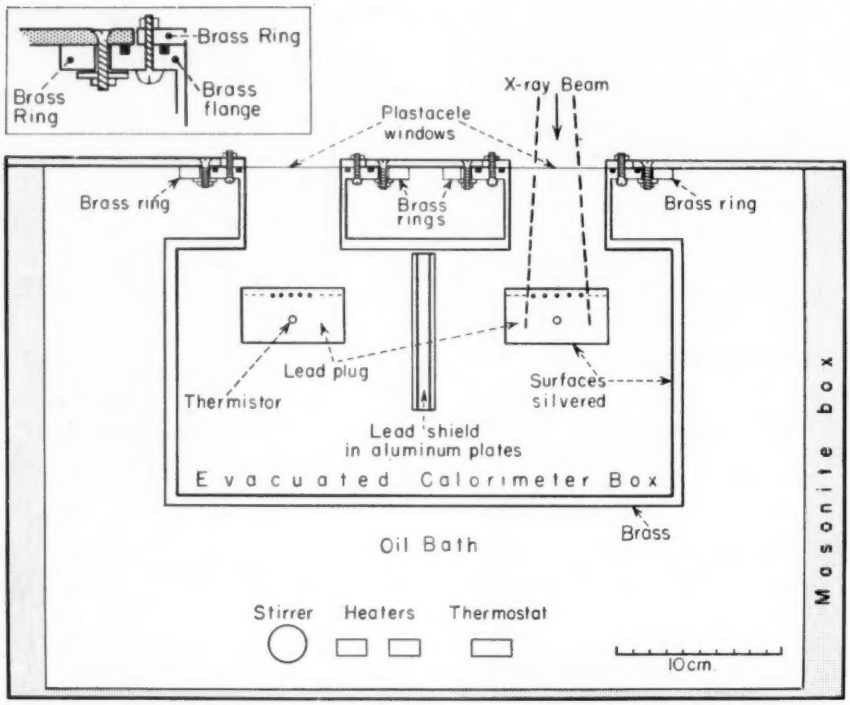

FIG. 6. Horizontal cross-section of calorimeter construction. All interior surfaces are silvered.

mounted inside a large masonite box which is filled with oil. The oil acts as a thermal "sink" so as to moderate the transmission of variations in room temperature to the calorimeter interior. A temperature control system maintains the oil bath at a constant temperature $\pm 0.1°$ C. so that the calorimeter can be used in different rooms without a delay to reach equilibrium.

The thermal losses of the calorimeter can be calculated approximately. The results of such calculations are reviewed briefly.

Radiation loss from the cylinders can be estimated from the relation:

$$E=\theta^2 AS(T_1^4-T_e^4) \qquad (2)$$

where E is the rate of energy loss in ergs /sec, θ is a coefficient of absorption taken in our case as 0.1, A is the surface area $=128$ cm^2, S is the Stefan Boltzman constant $=5.73\times10^{-5}$ erg/cm^2-degree^4-sec, T_1 is the cylinder temperature, and T_e is the equilibrium temperature of the calorimeter walls.

Conduction loss through the connecting wires and supporting strings can be estimated according to the relation:

$$\frac{dQ}{dt}=\frac{kA}{l}\Delta T \qquad (3)$$

where dQ/dt is the rate of energy loss in calories/sec, k is the thermal conductivity constant which for copper equals 0.92 calories/cm. degree sec, A is the area which for six No. 36 wires equals 10^{-3} cm^2, l is the length of the wires which was 10 cm., and T is the temperature difference over the wire.

Conduction loss through the air is also estimated from the same relation. The value of the thermal conductivity for air can be shown to be given by the following expression:

$$k=\frac{25\pi g}{64}\bar{v}Lc_v \text{ calories/cm sec degree} \qquad (4)$$

where g is the density of air, $\bar{v}$ is the mean molecular velocity, L is the mean free path, and c_v is the specific heat at constant volume. The molecular velocity can be

determined approximately from the relation $p=.33\,\bar{v}^2 g$ where p is the pressure. Since g varies directly with the pressure $\bar{v}$ will be independent of pressure to a first approximation. The value of $\bar{v}$ thus computed is 4.86×10^4 cm/sec.

The expression for L can be shown to be $L=1/(2^{1/2}n\pi\sigma^2)$ where n is the number of molecules/cm^3 and σ is the molecular diameter. At atmospheric pressure L is computed to be 6.1×10^{-6} cm. Since n is proportional to p, the value of L will vary inversely as the pressure, as long as its value is less than the dimensions of the container. The value of c_v is .17 calories /gram degree.

Since, in the expression for k, g varies directly as p, while L varies inversely as p, and the other terms are independent of p, k should remain constant until L reaches the dimensions of the container.[18] If the average distance between the cylinder surface and the container walls is taken as 5 cm., the thermal conductivity k would not be expected to decrease until the pressure was reduced to 10^{-3} mm. of Hg. In actual practice k was observed to decrease continuously with decreasing pressure which can probably be attributed to convection loss in the air.

The results of these calculations are summarized below for a one degree temperature differential:

Radiation	1.9×10^{-4} cal/sec
Conduction (wire)	1.0×10^{-4} cal/sec
Conduction (air)	1.5×10^{-3} cal/sec for pressure above 10^{-3} mm. Hg.

These values indicate that above 10^{-3} mm. Hg the conduction in air is the main avenue of heat loss.

The over-all value of k observed for the calorimeter at atmospheric pressure was 10^{-2} cal/deg sec, while at 10^{-2} mm. Hg it had dropped to 2.1×10^{-3} cal/deg sec. The latter value is not in disagreement with the calculated value for k. Evacuation to pressures above 10^{-3} mm. Hg is effective in reducing thermal loss due apparently to convection.

These losses are so small that the thermal loss correction during an experimental run is sufficiently small that each calorimetric determination has an experimental uncertainty of less than 1 per cent. Since other features of the experiment contain greater uncertainties, it is not important to reduce the value of k further. However, a diffusion pump has been added to examine the dependence of k on pressure.

ANALYSIS

The analysis of operation of the calorimeter and the experimental evaluation of the thermal loss coefficient k can be understood with the aid of Figure 7. The rate of heat loss dQ/dt of a body which exists at some temperature T relative to the temperature T_e of its environment can be represented according to Newton's cooling law as:

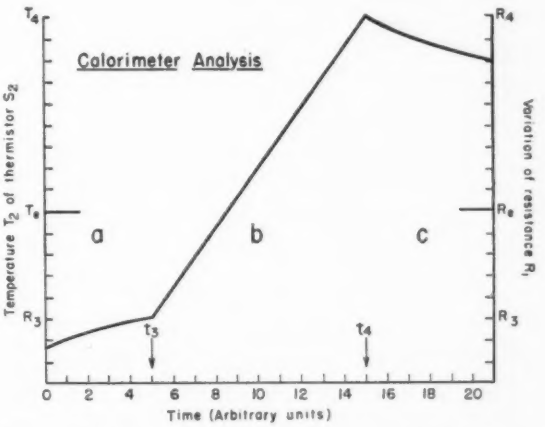

FIG. 7. This plot shows the temperature variation or bridge balancing resistance variation to be expected in a calorimeter before, during, and after absorption of energy at a uniform rate. All scales are arbitrary and the resistance and temperature scales are considered independently. Analysis is in the text.

$$\frac{dQ}{dt}=-McK(T-T_e) \qquad (5)$$

where dQ/dt is the change in calories/second, M is the mass of the body in grams, c is its specific heat in calories/gram-degree and K is in seconds^{-1} and is equal to k/Mc

where k is as defined above. Since $dQ = McdT$, equation (5) can be written in terms of the change of temperature dT of the body as:

$$\frac{dT}{dt} = -K(T - T_e). \tag{6}$$

The variation of the body temperature (T) as a function of time (t) is what is usually observed in a calorimeter. In Figure 7 the solid curve represents the observed temperature variation. In region a the asymptotic approach to equilibrium temperature can be written as

$$\left(\frac{dT}{dt}\right)_a = -K(T_a - T_e) \tag{7a}$$

and similarly in region c we have

$$\left(\frac{dT}{dt}\right)_c = -K(T_c - T_e). \tag{7b}$$

The constant K can be evaluated from the equation:

$$K = \frac{\left(\frac{dT}{dt}\right)_a - \left(\frac{dT}{dt}\right)_c}{T_c - T_a}. \tag{8}$$

In region b the observed rate of change of temperature $(dT/dt)_b$ is due to the influence of the external action we desire to measure $(dT/dt)_x$ and also to the thermal loss $-K(T - T_e)$;

$$\left(\frac{dT}{dt}\right)_b = \left(\frac{dT}{dt}\right)_x - K(T - T_e) \tag{9}$$

or

$$(dT)_x = \left(\frac{dT}{dt}\right)_b dt + K(T - T_e)dt. \tag{10}$$

This expression can be integrated from time t_3 to t_4 to determine the net change in temperature $(\Delta T)_x$ due to the influence of the external action in which we are interested:

$$(\Delta T)_x = \int_{t_3}^{t_4} \left(\frac{dT}{dt}\right)_b dt + K \int_{t_3}^{t_4} (T - T_e)dt. \tag{11}$$

If $(dT/dt)_b$ is linear the first integral is equal to $(T_4 - T_3)$ while the second integral can be shown to be equal to

$$\left(\frac{T_4}{2} + \frac{T_3}{2} - T_e\right)(t_4 - t_3),$$

and:

$$(\Delta T)_x = T_4 - T_3 + K(t_4 - t_3)\left(\frac{T_4 - T_3}{2} - T_e\right). \tag{12}$$

In our experiment the resistance required to balance a bridge is directly observed rather than the temperature. The foregoing analysis will be applied to the resistance measurements.

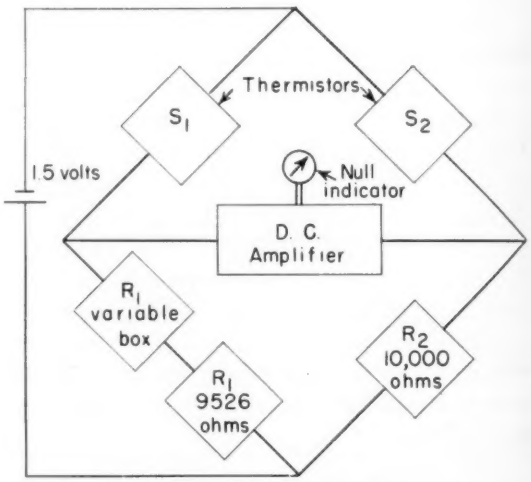

FIG. 8. Diagram of bridge circuit. Leeds and Northrup precision fixed resistors are used and the variable part of R is a special No. 4755-S Leeds and Northrup A.C. resistance box. All arms have about the same resistance. The chopper amplifier (Liston-Folb) input impedance matches that of the bridge. Amplified unbalance is indicated by the meter which serves as a null detector.

Each of the thermistors in the suspended cylinders is placed in opposing arms of a Wheatstone bridge as shown in Figure 8. The fixed resistors in the other arms are Leeds and Northrup manganin precision resistors. The variable resistance is a Leeds and Northrup box capable of adjustment over a range of 1,000 ohms in 0.01 ohm steps. A Liston-Folb chopper amplifier with an input impedance of 10,000 ohms amplifies the unbalance of the bridge. In

opposing arms of the bridge the effects of changes in environment temperature or energy dissipation by the bridge current in the thermistors tend to cancel.

In the balanced state the bridge resistances are related as:

$$R_1 = R_2 \frac{S_1}{S_2} \tag{13}$$

where S_1 and S_2 refer to the thermistor resistances.

The variables R_1, S_1, and S_2 are related as:

$$dR_1 = \frac{R_2}{S_2^2}(S_2 dS_1 - S_1 dS_2). \tag{14}$$

Since

$$S = S_0 e^{B(1/T - 1/T_0)}$$

and

$$\frac{dS_2}{dT_2} = -\frac{BS_2}{T_2^2} \tag{15}$$

$$dR_1 = BR_1\left(\frac{dT_2}{T_2^2} - \frac{dT_1}{T_1^2}\right), \tag{16}$$

where T_1 and T_2 refer to the thermistor temperatures. If, as in our case, $dS_1 = 0$, and $dT_1 = 0$:

$$\frac{dR_1}{dT_2} = \frac{BR_1}{T_2^2} = \frac{B}{T_2^2} \times \frac{R_2 S_1}{S_2}. \tag{17}$$

Since the dependence of S_1 and S_2 on temperature are similar, the ratio of S_1/S_2 would not be expected to vary. In fact, over a range of 1,000 ohms the ratio of S_1/S_2 remained constant between 1.007 and 1.009. The solid curve in Figure 9 is a plot of dR_1/dT_2 as a function of thermistor resistance S_2 using the experimentally measured values of B and R_1.

Since the variation of the resistance R_1 is what is actually observed experimentally, it is necessary to define its dependence on the temperature T_2 of the irradiated cylinder and thermistor. Over the resistance and temperature range employed in any experimental run, dR_1/dT_2 is essentially a constant. Since R_1 and S_2 are essentially equal and vary inversely, if $dR_1/dT_2 = m$, then $dS_2/dT_2 = -m$. m is a function of T_2 but is constant during each experiment. At $T = 300°$ Kelvin, $m = 384$ ohms/degree, which for a 10,000 ohm thermistor represents a negative temperature coefficient of resistivity of -3.84 per cent. We can now write:

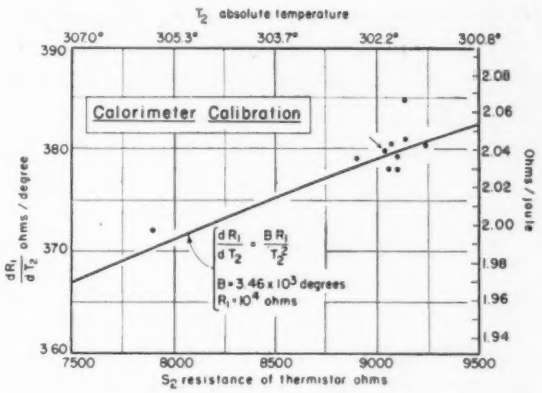

FIG. 9. Plot of dR_1/dT_2 versus S_2 and T_2. (dR_1/dT_2 is equal in magnitude to the negative temperature coefficient of resistivity of the thermistor S_2, and is proportional to the sensitivity of the calorimeter. The solid curve dR_1/dT_2 is based on the experimental determination of B in Figure 1. The calorimeter sensitivity is given by the right ordinate scale in ohms/joule as a function of S_2 and T_2. The experimental points are the results of the calibration runs such as represented in Figure 10 and are plotted against the right ordinate scale. Normalization of the ordinate scales is discussed in the text. Arrow indicates point obtained at atmospheric pressure.

$$\frac{dR_1}{dt} = \frac{dR_1}{dT_2}\cdot\frac{dT_2}{dt} = -mK(T_2 - T_e). \tag{18}$$

Over each experimental range we can also write:

$$R_1 = mT_2 + b, \quad \text{and} \quad R_e = mT_e + b$$

where T_2 and T_e are sufficiently close to have the same value of m and b during any experimental run. Substitution yields:

$$\frac{dR_1}{dt} = -K(R_1 - R_e) \tag{19}$$

which is identical with equation (6) for the dependence of temperature on time. We may employ the earlier analysis to

write for the net change in resistance due to the action of external roentgen rays:

$$(\Delta R)_x = R_4 - R_3 + K(t_4 - t_3)\left(\frac{R_4+R_3}{2} - R_e\right) \quad (20)$$

and for K:

$$K = \frac{\left(\frac{dR}{dt}\right)_a - \left(\frac{dR}{dt}\right)_c}{R_c - R_a} \quad (21)$$

all of which factors can be obtained from the plots of the experimental data. It can be shown that equations (20) and (21) are still valid when the effect of temperature drifts on the unirradiated cylinder are considered, and also for the case where the values of the resistance-temperature coefficient m are different for the two arms of the bridge.

EXPERIMENTAL

The calorimeter is calibrated by making experimental runs with a known amount of energy dissipated in the absorbing cylinder by the embedded heating coil. This coil is made of constantan and is soldered in a groove to establish good thermal contact. A specially low melting point solder is mixed for this prupose to avoid damage to the insulation. Copper leads are joined to the constantan inside the cylinder and brought out the lid through Kovar seals. The resistance of the heating element is precisely measured with a Wheatstone bridge. A Weston milliammeter calibrated with standard resistor, standard cell, and type K-2 potentiometer measure the current passing through the heating coil.

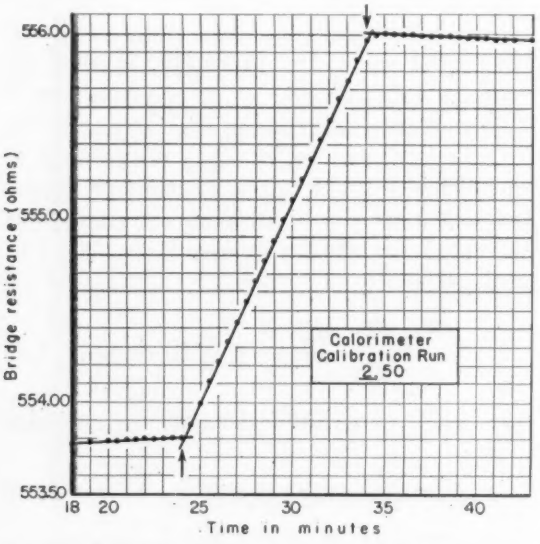

FIG. 10. Plot of typical data taken during a calibration run. The resistance of the variable resistance box at R required to keep the bridge balanced is plotted against time. The rapid linear rise between 24 and 34 minutes is produced by the dissipation of a measured amount of energy in the absorbing cylinder at a constant rate. The effect of the thermal losses on the net resistance change can be evaluated from the drift rate before and after heating. The notation 2, 50 refers to a data book.

Figure 10 displays a typical calibration "run" in which a measured amount of energy is dissipated at a constant rate over the same length of time used with external irradiations. During the cooling portions before and after the heating interval, the resistance R_1 can by interpolation be read to 0.001 ohm. A lag of approximately twelve seconds was consistently observed after the start of the heating current or irradiation and the commencing of a linear rate of temperature increase in the thermistor. The same lag was observed after the heating or irradiation ceased. The resistance of the thermistors is measured absolutely by an auxiliary Wheatstone bridge before and usually following each experimental "run." With the analytic procedure already described, experimental plots, such as Figure 10, are used to determine the net resistance change caused by the known dissipated energy. The sensitivity of the calorimeter can then be expressed in ohms/joule.

This sensitivity is proportional to the variation of dR_1/dT_2 with thermistor resistance (S_2) and temperature (T_2). Figure 9 contains a plot of dR_1/dT_2 versus S_2 computed from the experimentally determined values of B and R_1. The results of all of the calibration runs in ohms/joule have also been plotted on the same graph with the ordinate scale shown on the right border of the graph. The right ordinate scale is normalized with 2.40 ohms/joule cor-

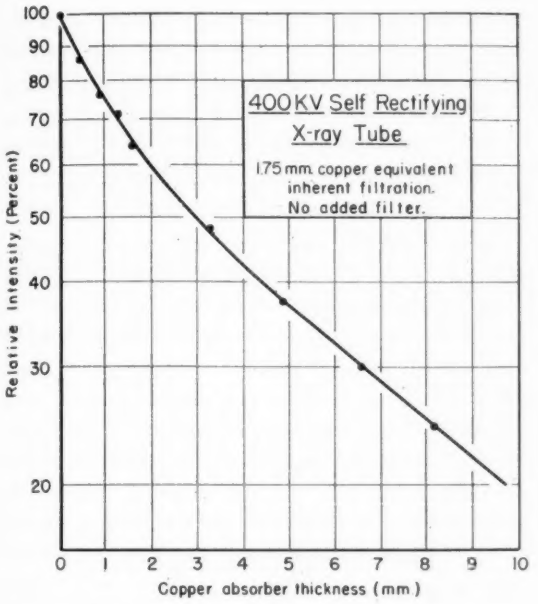

Fig. 11. Plot of transmission of the roentgen rays from the 400 kv. (peak) generator through indicated thickness of copper.

responding to 380 ohms/degree on the left ordinate scale. This normalization, which yields a reasonable agreement between the experimental calibration runs and the solid curve, corresponds to a heat capacity for the cylinder of 44.5 calories/degree. The value of the heat capacity calculated from the dimensions of the brass and lead components is 44.0 calories/degree which is good agreement considering uncertainties in the calculated value. The grouping of the results indicates the reproducibility of the method. One run was made with increased temperature and is not in disagreement with the solid curve. With the recently installed thermostatically controlled oil bath, a systematic investigation of this temperature dependence of sensitivity will be undertaken. For the experimental irradiation "runs" the solid curve calibration has been employed with the right ordinate scale.

A total of 9 "runs" was made with one of the absorbing cylinders exposed to the roentgen-ray beam from a 400 kv. General Electric "Maximar." This generator has an inherent filtration of 1.75 mm. copper equivalent. In this experiment no added filter was employed and the measured half-value thickness was 3.0 mm. of copper A plot of the absorption measurements made with a collimated beam from this generator is shown in Figure 11. This plot is the most complete simple manner of representing the quality of the beam employed. The diameter of the beam was 3.0 cm. at 50 cm. target skin distance, where the front surface of the irradiated cylinder was located.

A typical "run" is plotted in Figure 12. Ten minute irradiations were employed. Although most of the calibration "runs" were made in a constant temperature room, the therapy room temperature varied from day to day. A thermostatic heating control has been added to the oil bath since these data were taken. Table 1 contains the data of the irradiation experiments listed in order of increasing temperature. The second column gives the value of K calculated by equation (21). The third column lists the net changes in resistance observed ac-

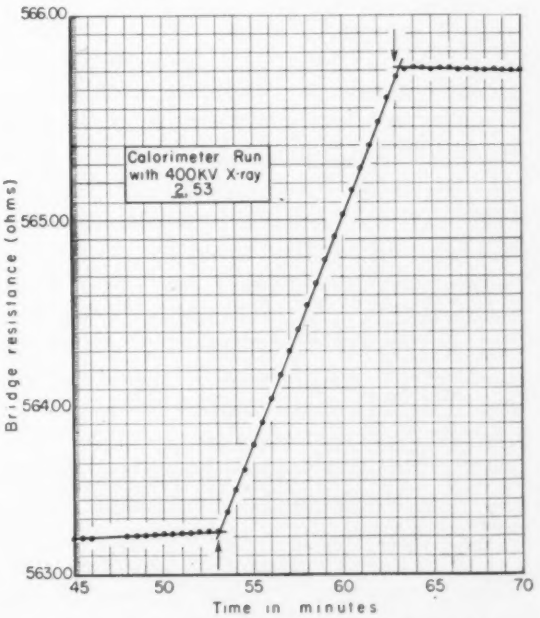

Fig. 12. Plot of typical data taken before, during, and after exposure of calorimeter to the collimated roentgen rays of a 400 kv. (peak) generator. Analysis is same as calibration run in figure 10. The notation 2, 53 refers to a data book.

cording to equation (20). Column 4 lists the equivalent number of joules required to produce the observed resistance change. The calibration curve in Figure 9 is employed for this conversion. The average value is 1.21 joules with a probable error of ±.01. With an increased number of runs at a controlled constant temperature, the precision of this determination can be increased even further.

TABLE I

400 KV. CALORIMETER RUNS

Thermistor Resistance S_2 (ohms)	Thermal Time Constant K (seconds^{-1})	Net Resistance Change $(\Delta R)x$ (ohms)	Joules Required to Produce Same Net Change (joules)
9120	$.92\times10^{-4}$	2.46	1.21
8870	2.02×10^{-4}	2.41	1.19
8820	$.57\times10^{-4}$	2.46	1.21
8790	$.64\times10^{-4}$	2.44	1.20
8750	$.57\times10^{-4}$	2.43	1.20
8700	$.54\times10^{-4}$	2.47	1.22
8400	$.70\times10^{-4}$	2.45	1.21
8370	$.59\times10^{-4}$	2.48	1.23
8340	$.37\times10^{-4}$	2.48	1.23

The output in roentgens was determined with a series of measurements with three different Victoreen thimbles which had been recently calibrated by the Victoreen Company. After pressure and temperature corrections the output at 50 cm. in air was 60.5 roentgens/minute with a probable error of all readings of slightly less than 1 per cent. Densitometer measurements of the intensity distribution over the area of the beam indicated that the average intensity was 98.2 per cent of that in the center. The final corrected output figure was 59.4 roentgens/minute and .99 roentgens per second.

The resulting energy flux based on 1.21 joules dissipated by a roentgen-ray beam 3 cm. in diameter at the rate of .99 r/sec over an interval of 600 seconds is 2880 ± 90 ergs/cm²-roentgen. The uncertainty contains the probable error in the calibration and radiation runs together with 1 per cent uncertainties in the roentgen rate determination and in the area of the beam. Uncertainties in the time of 600 seconds appear in the probable error of the radiation runs.

It is necessary to determine what proportion of the energy in the beam is backscattered from the front surface of the cylinder relative to the energy absorbed. Ionization measurements were made for this purpose. The energy dissipated in the air in front of the cylinder is evaluated by collecting the ionization produced in the air in front of the cylinder position with and without the cylinder. This difference ionization is that produced by the backscattered electrons and soft roentgen rays.

A large transmission ion chamber with thin (.003 inch) aquadag coated nylon walls was employed. The chamber contained three parallel conducting surfaces, the outer two at ground potential while the inner one was the collecting surface at 536 volts below ground. The plate separation was 1.25 cm. and the field of 430 volts/cm. was sufficient for saturation at 1 r/sec. The diameter of the collecting area was 12 cm. The ionization produced in the air in this chamber was measured at successive distances from the cylinder surface towards the target. A small polystyrene chamber with a plate spacing of 0.89 mm. was also used for measurements close to the surface of the cylinder. The wall thickness of this chamber (.5 mm. polystyrene, density = 1.38 grams/cm²), was sufficient for equilibrium for these unfiltered 400 kv. roentgen rays. The total ionization measured by the small chamber integrated over the first 2.5 cm. distance in front of the cylinder agreed closely with the ionization collected by the large chamber in this location. The results of these ionization measuremen s plotted in esu/cm-sec. as a function of distance from the cylinder surface are shown in Figure 13 for both a brass surface and a lead surface. The large chamber diameter (12 cm.) is adequate to collect all ionization regardless of the angle of scattering. The area under the plots in Figure 13 yields the total esu/sec collected.

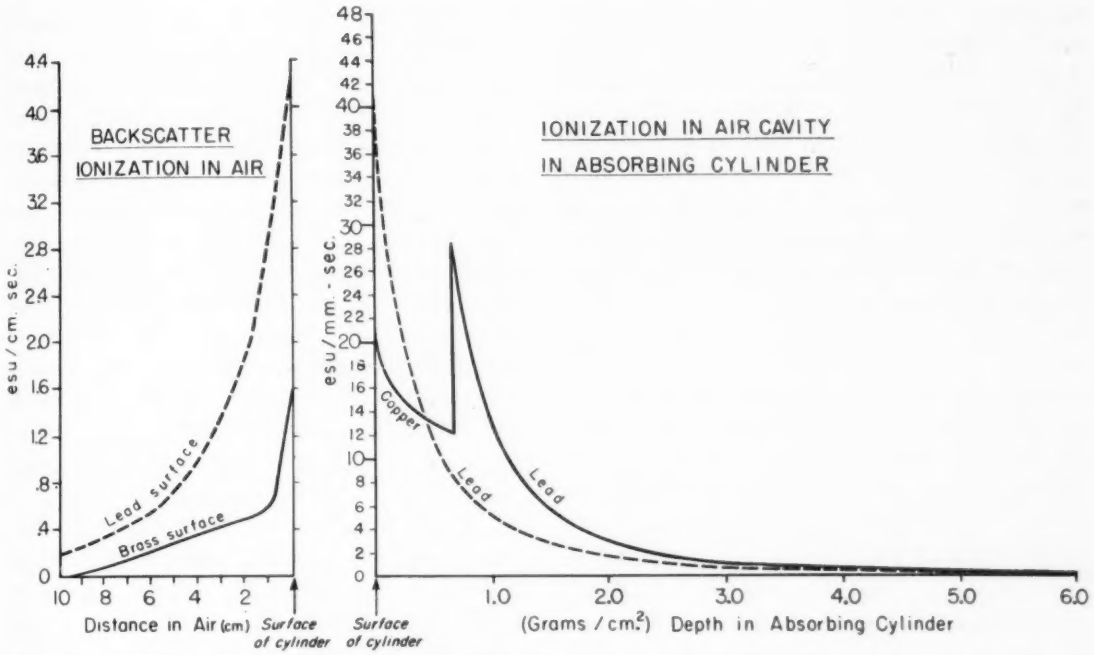

FIG. 13. The left graph is a plot of ionization in air by the radiation backscattered from the absorbing cylinder. These data can be analyzed to evaluate the amount of energy lost by backscatter. The right graph is a plot of the ionization observed inside the absorber obtained with the ionization chamber in Figures 14 and 15. These data can be used to estimate the energy absorbed in the absorbing cylinder.

This can be converted into ion pairs produced per cm²-roentgen of the incident beam. Using 32.5 electron volts as the average energy required to produce one ion pair in air, the energy dissipated by backscatter in the air per cm²-roentgen of the incident beam is calculated. For the brass surface the energy dissipated in air is 0.053 ergs/cm²-roentgen and for a lead surface the amount is 0.196 ergs/cm²-roentgen.

The energy dissipated in the absorbing cylinder is also estimated by ionization measurements. To do this, a lead ionization chamber (Fig. 14 and 15) was constructed. The depth of this chamber was 0.5 mm. as determined by a polystyrene spacer. This dimension is small compared to the ranges of the secondary electrons in air traversing the chamber. The thickness of the front surface towards the target is varied by adding or substracting lead sheets. The distance between the target and front surface was maintained at 50 cm. The chamber diameter is sufficiently large to collect all ionization produced by the beam in the absorbing cylinder. This chamber connected directly to a preamplifier which fed into an amplifier used in previous ionization measurements.[12] Figure 13 shows the data obtained with this chamber corrected to standard pressure and temperature. The number of electrostatic units collected per second are plotted versus the grams/cm² of front wall absorber. Since the actual calorimeter cylinder had a brass jacket, copper sheets were used to simulate the absorption near the surface. A plot is also shown obtained with only lead sheets.

A relation between air cavity ionization and energy dissipation in the surrounding wall has been developed by Bragg[2] and Gray.[6] This relation is stated as:

$$E_v = \rho W \mathcal{J}_v \qquad (22)$$

where E_v is the energy dissipated per unit volume in the wall, ρ is the ratio of the energy loss per cm. path of the secondary electrons in the wall and in air, W is the average energy expended by an electron

per ion pair formed, and $\mathcal{J}_v$ is the ionization per unit volume observed in the air cavity. The air cavity dimensions are required to be small with respect to the range of the secondary electrons in air. If the energy absorbed per unit volume in the absorbing material is integrated over the volume, the total energy E absorbed is obtained. The relation above can be restated for the total energy absorbed as:

$$E = \rho W \mathcal{J} \tag{23}$$

where $\mathcal{J}$ is the total ionization to be collected if the cavity chamber were moved throughout the absorbing volume. The value of $\mathcal{J}$ in total esu collected per second can be evaluated from the area under the solid curve in Figure 13. The value of W is taken as 32.5 electron volts.[27] The quantity ρ can be defined as:

$$\rho = \frac{(S)_{\text{medium}}}{(S)_{\text{air}}} = \frac{\left(-\frac{dE}{dx}\right)_{\text{medium}}}{\left(-\frac{dE}{dx}\right)_{\text{air}}} = {}_eS \frac{n_{\text{medium}}}{n_{\text{air}}} \tag{24}$$

where S and $-(dE/dx)$ refer to the stopping power in energy loss per unit path length, and ${}_eS$ is the stopping power per electron of the medium relative to air. The equation, due to Bethe[5]

$$-\frac{dE}{dx} = \frac{2\pi n e^4}{mv^2}\left[\frac{\ln mv^2T}{2I^2(1-\beta^2)} - \ln 2(2\sqrt{1-\beta^2}-1+\beta^2)+1-\beta^2\right] \tag{25}$$

where n is electron density, e is the electronic charge, m the electron rest mass, v the velocity of the incident secondary electron, T its relativistic kinetic energy, and I the ionization potential of the atoms, was used to calculate S. I is taken as 13.5 Z, where Z is the atomic number. Density polarization effects do not have to be considered for electrons of these energies.[7,13] Though calculated values of S vary rapidly with energy for electrons below 400 kv., the ratios ${}_eS$ are not strongly energy-dependent. Calulated values of S for 100 kv. electrons in various materials relative to air are: argon .93, copper .77, gold .66, and lead .65. The corresponding values of ρ are 1.15, 4880, 7880, and 4500, in the same order. Lead has an anomalously low electron density and has essentially the same ρ as copper. For 10 kv. electrons the calculated values of S are about 5 times greater but the values of ρ decrease only about 20 per cent and are thus not greatly energy dependent. The calculated value of ρ will depend somewhat on the form of the energy-loss equation employed, and the values used for the ionization potentials I. All energies of secondary electrons are present below 400 kv. which adds to the difficulty in making the proper choice of ρ. For the purpose of our estimate of the relative proportion of the energy absorbed to that backscattered, a precise value of ρ is unimportant. An intermediate value of ρ is used; for the copper region, 4200; and for lead, 4100. The value for the copper region of the absorber contains an allowance for the fact that the front surface of the chamber is copper while the rear collecting surface is always lead. The resulting values for E_A are about 3400 ergs/cm²-roentgen for the combination copper and lead ionization chamber phantom and 2500 ergs/cm²-roentgen for the all-lead chamber. Despite the divergence in these estimates, it is certain that the energy dissipated in the air by backscatter of 0.053 and 0.196 ergs /cm²-roentgen for the two chambers, respectively, is a negligible fraction of the total absorbed energy. Since the volume of the ion chambers is known to a few per cent, the divergence in the absorbed energy estimates indicates either a failure of equation (22) for high atomic numbers, or that the effective secondary electron energies are not the same in copper and lead. Since the accuracy of the calorimetric determination permits an absolute test of Gray's relation, carbon and aluminum ion chambers similar to that in Figure 14 have been constructed. Gray reported evidence that

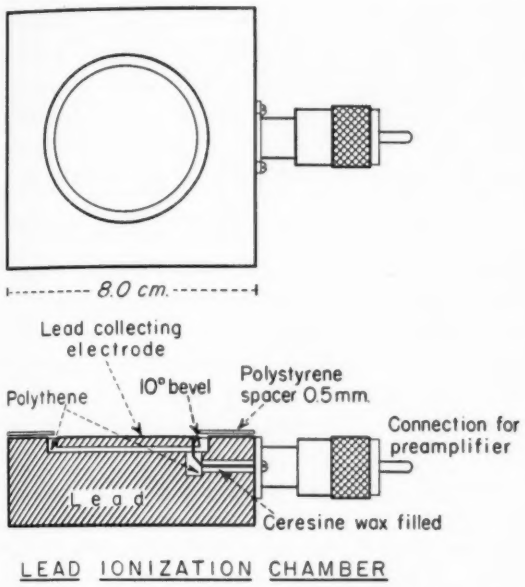

Fig. 14. Construction of cavity ionization chamber used to determine the distribution of energy in the absorbing cylinder.

the relation was not absolute for high atomic numbers but it is believed absolute for aluminum and carbon.

These ionization measurements were made primarily to determine the relative magnitude of energy loss due to backscatter and proved satisfactory for this purpose. Since this loss correction proves negligible, the final value remains at 2880 ± 90 ergs/cm^2-roentgen.

22.5 mev. Roentgen Rays. An improved version of our earlier calorimeter[15] was employed to measure the output of our betatron. The roentgen rays have a maximum energy of 22.5 mev. The beam is filtered by 3.5 grams/cm^2 of carbon and has a total absorption coefficient in water of approximately .026 cm^{-1} which corresponds to the absorption of 7.5 mev. homogeneous roentgen rays. The procedure is the same as for the measurements with the 400 kv. generator just described. The details and conclusions of our preliminary experiment have been described in the literature.[15,16] The energy flux is measured in terms of the reading of one 25 r Victoreen thimble chamber centered in an 8 cm. lucite cube, and in terms of the esu/cm^3 collected in a small air cavity ion chamber centered in an 8 cm. cube of water. This latter measurement can be converted into roentgens according to the convention outlined by Mayneord.[20] A correction of 0.7 per cent is necessary for energy loss by neutrons in photo-disintegration reactions. The absorbing cylinders are 9 cm. in diameter and 13 cm. long and consist of lead jacketed in silver-plated brass shells. Calibration heating coils and 100,000 ohm thermistors are mounted in each cylinder. The dimensions of the cylinders are made large in order to insure total absorption of the beam. Two different diameters of the roentgen-ray beam were employed, 2.5 cm. and 5.0 cm. in diameter. The results for the 22.5 mev. (peak) roentgen-ray beam are:

5700 ± 250 ergs/cm.^{2}—roentgen (Victoreen), the roentgen being the reading of Victoreen thimble chamber centered in an 8 cm. lucite cube;

5500 ± 250 ergs/cm.^{2}—esu/cc., the esu/cc. collected in a small air cavity centered in an 8 cm. cube of water;

5600 ± 250 ergs/cm.^{2}—roentgen (M), the roentgen determined according to Mayneord's convention;

5350 ± 250 ergs/cm.^{2}—roentgen, the roentgen determined from the depth dose curve with the requirement that the absorbed energy equal the dissipated energy.[16]

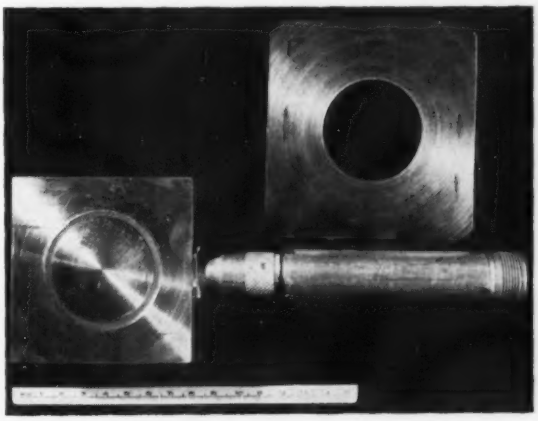

Fig. 15. Photograph of the ionization chamber in Figure 14. Sheets of lead or copper are added above the cavity to vary the wall thickness. Polystyrene spacer is shown separately.

The first two values of the experimental results are stated in terms of common experimental methods used to measure the ionization produced by the beam. They can be compared with theoretical calculations,[9] and also with experiments with a Lax chamber by McElhinney and Siewers.[17] The last two values have been corrected for the relative ionization in water and air-wall chambers, and should presumably be in terms of the true roentgen. These latter values can be compared with Mayneord's theoretical calculations.[21]

It must be emphasized that experimentally all the energy in the roentgen-ray beam must be absorbed if the accuracy possible with the calorimetric method is to be realized. In our first preliminary experiment in the application of calorimetric methods to a roentgen-ray beam, small absorbing cylinders were used which did not totally absorb the beam. Corrections based on relative ionization distributions in lead were used to compensate for the amount of energy not absorbed. Later experiments reported here with large cylinders demonstrate that these earlier corrections were not successful. The use of cylinders large enough to essentially absorb the total beam is feasible because of the sensitivity and reliability of these calorimetric techniques.

Comparison with Theory. Since the spectra of these 400 kv. and 22.5 mev. generators are continuous, a comparison with theory must weight the theoretical flux values according to the generator spectral distributions. If the theoretical energy flux E_A (E) in ergs/cm²-roentgen is calculated as a function of roentgen-ray energy E, and the intensity per energy interval is $I(E)$, then the mean value of the theoretical flux E is given by the relation:

$$\bar{E}_A = \frac{\int_0^{E_{\max}} I(E) E_A(E) dE}{\int_0^{E_{\max}} I(E) dE} . \qquad (26)$$

Under equilibrium conditions it can be shown theoretically that the definition of the roentgen is equivalent to the absorption of 83.7 ergs/gram-roentgen in air, assuming that an average of 32.5 electron volts is dissipated per ion pair formed. The energy flux E_A in ergs/cm²-roentgen can be related to this factor as follows:

$$E_A(\tau_a + \mu_a + \pi_a) = 83.7g \qquad (27)$$

where τ_a, μ_a, and π_a are the real absorption coefficients for the photoelectric, scattering, and pair production processes, respectively, and g is the density of air. The assumption that only the real part of the coefficient is effective is probably not strictly valid since some of the scattered quanta suffer absorption and contribute to the absorbed energy. The coefficients for air are well known, permitting a calculation of E_A as a function of energy. Mayneord[21] had calculated $E_A(E)$ in this manner and his plotted results have been used in equation (26) above.

For the 400 kv. generator the empirical expression by Kulenkampff[11] was used to calculate the initial intensity distribution. This spectrum was corrected for the absorption in 1.75 mm. copper to get the transmitted distribution $I(E)$. For 400 kv. roentgen rays $\bar{E}_A$ was calculated by numerical integration of equation (26) to be 3000 ergs/cm²-roentgen. This is in reasonable agreement with the experimental value of 2880 ± 90 ergs/cm²-roentgen. This comparison is not greatly affected if other spectral distributions are assumed.

For the 22.5 mev. (peak) roentgen rays the Schiff[25] theoretical spectrum was employed. This is modified for the absorption in the donut (procelain) wall and in the carbon filter. The result was $\bar{E}_A = 5200$ ergs/cm²-roentgen for Mayneord's graph of $E_A(E)$ and $\bar{E}_A = 5400$ ergs/cm²-roentgen when the values of $E_A(E)$ given by Mayneord[21] based on the work of Streib, Fowler and Lauritsen were employed. Considering the uncertainties in the conventions used to represent the experimental ionization determinations in terms of the roentgen, the experimental and theoretical values are in reasonable agreement.

The experimental value of 5700 ± 250 ergs/sq.cm.-roentgen (Victoreen in lucite) can be compared with a thoretical calculation of $E_A(E)$ in terms of a Victoreen thimble chamber in an infinite block of lucite.[9] This calculation utilizes equation (27) with the absorption coefficients of lucite rather than air. Numerical integration of equation (26) using their graph for $E_A(E)$ and modified for the attenuation in our finite lucite block, yielded $\bar{E}_A = 5800$ ergs/sq.cm.-roentgen (Victoreen in lucite). This value also agrees closely with the measurement of McElhinney and Siewers using a Lax chamber.[17]

The calorimetric part of the determinations described in this report are more accurate than our calibration of the generator output in roentgens, or knowledge of the area of the beam. Though thermostatic control of the oil bath, better vacuum, and improved technique can still further increase the precision of these calorimetric measurements, the over-all accuracy of the comparison with the roentgen is limited by the non-calorimetric part of the experiment whose accuracy can also be improved. Since the measurements here described with roentgen-ray beams of widely different energies have demonstrated the sensitivity and practicality of this method, it is intended to extend these flux measurements to various energies between 50 kv. and 25 mev. under varying conditions of filtration.*

Energy Dose. Precise determination of the ionization distributions produced in water by the radiation from these generators, together with these flux measurements permits an accurate experimental evaluation of the ergs absorbed per gram-roentgen.[16] This, in turn, will make it possible for the radiologist to know what factor to use to convert the percentages on his depth dose and isodose plots into actual ergs absorbed in water.

Examples of this application of these flux determinations are shown in Figures 17 and 18. The energy absorbed in water in ergs/gram is related to the incident energy flux in the following manner when the absorbed energy is required to be equal to the dissipated energy:

$$Eg(x) = E_A \frac{\mu\mu_s}{\mu_s - \mu} \left(\frac{S}{S+x}\right)^2 (e^{-\mu x} - e^{-\mu_s x}). \quad (28)$$

μ and μ_s are effective coefficients of the absorption of the primary radiation and secondary electrons, respectively, as determined from analysis of depth-dose distributions.[12] $Eg(x)$ is the energy absorbed at any depth x in ergs/gram per unit time, E_A is the incident flux in ergs/cm^2 per unit time, $\mu = .0235$ cm^{-1} for a large field for 22.5 mev. roentgen rays, and $\mu_s = .77$ cm^{-1}. This relation assumes that the distribution of ionization in a body is directly proportional to the distribution of dissipated energy. This proportionality should be constant beyond equilibrium depths in the body according to Gray's relation. In the surface region before equilibrium conditions exist, this proportionality is not necessarily constant. It is not believed that the variation in this region is sufficiently large to greatly affect the validity of equation (28). Figure 17 is a plot of equation (28) for $S = 80$ cm. and 22.5 mev. (peak) roentgen rays. Figure 18 is a plot of the energy ab-

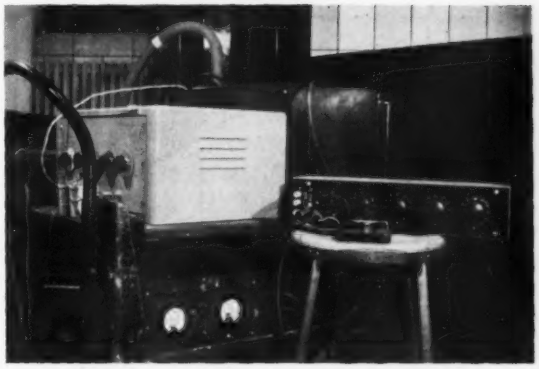

Fig. 16. Photograph of apparatus in position in front of the 400 kv. (peak) generator. A special collimator collimated the beam and mechanically connected the calorimeter and generator. The apparatus is portable. Resistance box and meter on stool are in control room during irradiation.

* These measurements are in progress in the Department of Physics, Sloan-Kettering Institute, Memorial Center, where an improved calorimeter has been constructed with the assistance of the Atomic Energy Commission Contract AT (30-1)-1451.

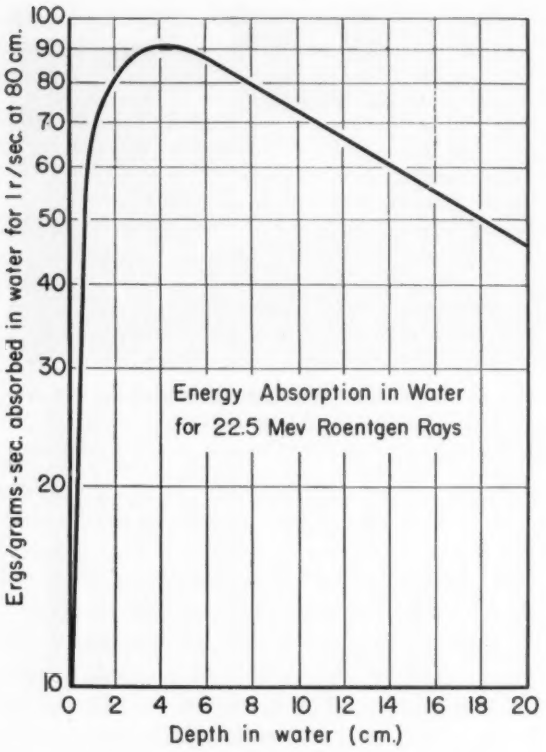

FIG. 17. The energy absorbed in ergs/gram-sec in water is plotted according to equation (27) as a function of depth in a water body.

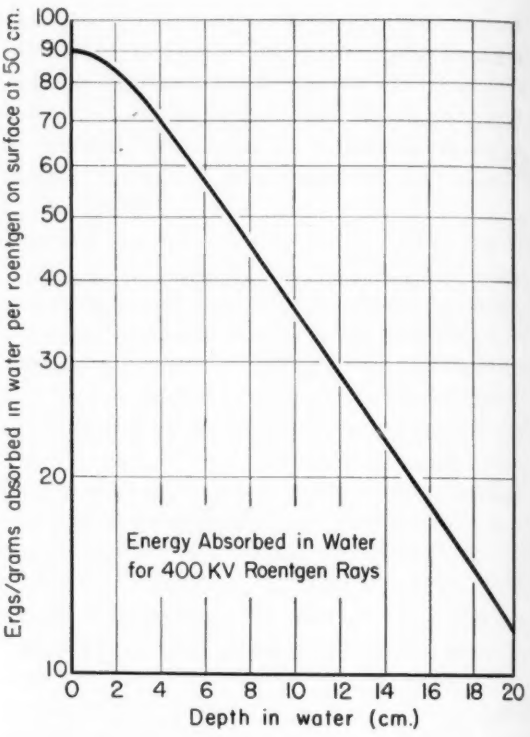

FIG. 18. The energy absorbed in water in ergs/gram per roentgen on the surface is plotted as determined from the energy flux determination together with the depth dose curve for the central axis of a 150 cm² field.

sorbed per gram of water for the 400 kv. roentgen rays as determined from our energy flux measurement and a depth dose curve. These figures (Fig. 17 and 18) permit convenient evaluation of the energy absorbed at any depth in a body.

In conclusion, it has been shown that it is technically possible to make the energy flux in ergs/cm² the fundamental calibration of the output of a roentgen-ray generator. Such a provision has definite advantages, particularly in the energy region above 1 mev. where the roentgen cannot be measured directly.

SUMMARY

This report has covered the following subjects:

1. The importance and significance of the distinction between intensity and dose has been emphasized.

2. A calorimetric method of sufficiently high sensitivity to measure the output (beam intensity) of any roentgen-ray generator in use has been described.

3. This method has been applied to measure the output of the two roentgen-ray generators with these results:

400 kv. (peak) (3.0 mm. copper half-value layer): 2880 ± 90 ergs/cm²—roentgen;

22.5 mev. (peak) (3.5 gram/cm² carbon filter): 5700 ± 250 ergs/cm²—roentgen (Victoreen in lucite).

4. The combination of flux measurements and ionization distribution measurements has been applied to obtain plots of the energy dose in ergs per gram as a function of depth in the irradiated body.

5. It is suggested that such a calorimetric method of flux determination as developed here can be made the fundamental calibration of the output of roentgen-ray genera-

tors, particularly for generators above 1 mev.

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DISCUSSION

Dr. H. W. Koch, Washington, D. C. The calorimetric method of measuring roentgen-ray intensities which has just been described is a very precise experiment and a very timely one. It comes at a time when there has just been international agreement to shift the emphasis from measuring intensities in terms of roentgens per second to measuring them in terms of energy units. This applies to measurements of energy flux in ergs per centimeter squared per second and of energy dissipated at a point in a medium in ergs per centimeter cubed. It is timely also because there have been no precise ways of measuring energy flux of high energy roentgen rays prior to the work of the group at the University of Illinois College of Medicine.

The results that are of particular interest to workers in the betatron field were those measurements made with the 22 million volt beta-

tron. The results were reported in units of ergs per centimeter squared per ion pairs per centimeter cubed where the number of ion pairs per centimeter cubed measured was qualified by the type and thickness of material surrounding the measuring device. Dr. Laughlin quoted results for a Victoreen r-meter thimble located either in an 8 centimeter cube of water or of lucite. Also, interpretations of the roentgen according to conventions proposed by Dr. Mayneord and another by Dr. Laughlin were cited.

The very fact that there are at least two interpretations of what a roentgen means at high energies indicates the real wisdom of trying to provide dose standards in terms of energy units rather than in terms of roentgen units. Another convincing point regarding energy units was the fact, drawn from this paper, that the least precision was obtained from the measurements of the roentgen while the measurements of energy in the beam in ergs per centimeter squared were quite precise.

Their work should serve as a real challenge to those who are interested in making intensity measurements. We hope to accept this challenge at the betatron laboratory at the National Bureau of Standards. We would like to correlate intensity measurements made with a calorimeter with measurements made by other methods. For example, we are attempting to make measurements made by a calorimeter consistent with those made by ionization chambers and by roentgen-ray spectrometers. In a roentgen-ray spectrometer, two of which I have described at these sessions earlier, one can measure the individual roentgen-ray energies and can sum these in order to determine energy flux. These flux values must be consistent and will have to check those obtained with a calorimeter.

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Fifty-fourth Annual Meeting: Netherland Plaza Hotel, Cincinnati, Ohio, September 29–October 2, 1953.

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Thirty-sixth Annual Meeting: The Homestead, Hot Springs, Virginia, March 14, 15 and 16, 1954.

EDITORIALS

THE ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fifty-fourth Annual Meeting of the American Roentgen Ray Society will be held at the Netherland Plaza Hotel, Cincinnati, Ohio, September 29 to October 2, 1953. The Society returns to Cincinnati well knowing that it will be splendidly housed in the Netherland Plaza; its facilities are such as to assure an excellent meeting and if further evidence were needed one has only to turn elsewhere in this issue of the JOURNAL to see President-elect Weber's noteworthy program which he has arranged for this year's meeting. The program is an excellent one, covering as it does a wide range of papers on the diagnostic aspects of roentgenology; in addition, there is a group of papers dealing with the therapeutic aspects of radiology; and another group on the always timely subject of protective measures in radiation exposure, and an excellent symposium has been arranged dealing with the practical consideration of mitral stenosis, covering the clinical aspects and physiologic changes, surgical observations and the roentgenologic contributions.

On Tuesday evening the Caldwell Lecture will be given. Dr. Paul C. Hodges, Professor and Chief of the Department of Radiology, University of Chicago, Chicago, Illinois, has been chosen to give this year's Caldwell Lecture, and he has selected as his topic "Roentgen Diagnosis of Skeletal Disease." Dr. Weber and his committee have made a wise decision in the selection of so eminent a radiologist and scientific investigator as Dr. Hodges, and his topic of discussion should prove profitable and may be anticipated with pleasure.

Under the direction of Dr. B. R. Kirklin who has charge of the Instruction Courses at the forthcoming meeting, there has been selected not only a distinguished faculty but a comprehensive course of lectures which should make a special appeal to all the members and guests. Elsewhere in this issue will be found the detailed information concerning the Instruction Courses, and it is urged that those who anticipate taking the courses register at once; otherwise they may find the courses of their choice filled by previous applicants. President-elect Weber has so arranged his program that the Instruction Courses will begin this year at three o'clock in the afternoon.

The Netherland Plaza Exhibits Halls are admirably suited to house the Scientific and Commercial Exhibits because they are so close to the Pavillon Caprice where the scientific sessions of the meeting will be held. The Scientific Exhibit promises to be an exceptional one this year, and as usual the Commercial Exhibitors are showing great interest in the forthcoming meeting realizing as they do that there will be ample space for display of their newer instruments and apparatus for roentgenological and ancillary fields. Included in the exhibits will be the latest precision instruments for use with radioisotopes, and there will also be found the publishers of scientific books dealing with radiology and medicine.

In the preparation and arrangement of this year's program, President-elect Weber and his committee have not overlooked the social aspects of the meeting. The Annual Gulf Tournament for the Willis F. Manges trophy will be held on Monday, September 28, at the Kenwood Country Club and the annual Golf Dinner will be held at the same place after the tournament. Those

who have been privileged to play the beautiful golf course at the Kenwood Country Club will be pleased that the tournament is again to be held at that delightful spot.

On Thursday evening will be held the Annual Banquet in the Pavillon Caprice and the banquet will be preceded by a cocktail hour. At the banquet will be presented the awards for the Scientific Exhibits at this year's meeting.

The excellent facilities of the Netherland Plaza are too well known to the members of the Society and their guests to need any further discussion but it is urged that those who plan to attend this meeting make their reservations at as early a date as is possible for them to do so. The hotel facilities in Cincinnati are ample to care for all those who anticipate attending the meeting providing they make their reservations early.

Request for reservations should be made by writing Mr. Wilbur Wright, Reservation Manager, Netherland Plaza Hotel, Cincinnati, Ohio.

EDWARD HOLMAN SKINNER
1881-1953

THE death of Dr. Edward Holman Skinner on January 11, 1953, takes from our midst an interesting, arresting, and vibrant personality. A man of unquenchable energy, he was throughout his professional life in the forefront of movements dedicated to scientific progress, to the elevation of medical standards, and to the great task of making the profession aware of its obligations to the public.

Dr. Skinner was born in 1881 in Milwaukee, Wisconsin, but spent most of his youth in St. Louis. He received the degree of M.D. from St. Louis University in 1904, and after doing postgraduate work, including a year abroad spent mostly in the study of roentgenology, he came to Kansas City in 1906 to begin the practice of his profes-

sion. He can well be classed with the pioneers of roentgenology since he began as a specialist in this field only ten years after Röntgen's epochal discovery.

Dr. Skinner cherished from his mother, who was also a physician, an axiom—"Son, don't wait for your ship to come in; go out in a tug and meet it." This axiom became his *leitmotif*, and he applied it particularly to the problems of medicine which arose with the new health needs in these changing times.

Almost immediately after his arrival in Kansas City, Dr. Skinner became very active in child welfare and in related aspects of community medicine, including summer camps for babies. In the course of this work, he became acquainted with Miss Irene Stowell, and they were married in 1912. She survives him.

One of his earliest and most lasting loves was that of books. In the time of his arrival in Kansas City, the Jackson County Medical Society had no library. In 1911, largely through his own individual efforts, a library club was formed, which acquired books and, after assembling a sizable library, presented it to the Medical Society. Dr. Skinner himself began early in his career to assemble important books and monographs on radiology. As a pioneer, he saw the contemporary publications soon become classics in their field. He continued throughout his life to purchase important publications on radiology and assembled them into a notable library of books on electricity, roentgen rays and radium, which he presented to the Library of the History of Medicine of the University of Kansas in 1951.

Always active in medical organizations and in all educational efforts of the medical profession, Dr. Skinner was past president of the American Radium Society, the American Roentgen Ray Society, and the American College of Radiology. In 1941, he received the Janeway Award from the American Radium Society, and, with Dr. Arthur C. Christie, he had been awarded in 1952 the gold medal of the American College of Radiology for leadership in socio-economic aspects of medical care. He was the driving force in the formation of the Kansas City Southwest Clinical Society in 1922, was its first president, and served until his death as editor of its Journal. This organization looks back on a thirty year successful career, attracting more than a thousand physicians each year to its meetings and serving as a model for numerous similar organizations which have sprung up throughout the Middle West.

Dr. Skinner loved to acquire knowledge and loved equally well to impart it. In 1932, he established a circuit of ten smaller cities in Missouri and Kansas, where he held regular clinics demonstrating particularly the value of the roentgen rays in the diagnosis and treatment of cancer. This arduous work, which required eight days out of each month, he carried on for twenty years, up to the date of his death.

His services in advancing postgraduate education, broadening the base of medical care, and improving physician-patient relationships were outstanding. Yet, he was a bitter opponent of federal insurance and sickness plans and was an active member of the National Physicians Committee, which unquestionably played an important role in defeating such legislation.

Dr. Skinner was associated with the Department of Roentgenology in St. Luke's Hospital for thirty years, St. Mary's Hospital for twenty-eight years, Kansas City General Hospital for seventeen years. In 1940, he became a lecturer in the Department of the History of Medicine at the University of Kansas and later was appointed a lecturer in the Department of Radiology. He will be long remembered by students as an interesting, clear, and forceful speaker and teacher.

Ned Skinner will be missed by a large circle of friends both professional and lay. He was an aggressive champion of all he considered right and proper, an unsparing critic of certain delinquencies of medical practice, but he was always generous and fair-minded and never based his judgment on bias or prejudice.

Ralph H. Major, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Pa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Sept. 29–Oct. 2, 1953.

American Radium Society
Secretary, Dr. R. E. Fricke, Mayo Clinic, Rochester, Minn. Annual meeting: The Homestead, Hot Springs, Virginia, March 14–16, 1954.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 713 E. Genesee St., Syracuse 2, N. Y. Annual meeting: Palmer House, Chicago, Ill. Dec. 13–18, 1953.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Illinois.

Section on Radiology, American Medical Association
Secretary, Dr. T. Leucutia, Harper Hospital, Detroit 1, Mich. Annual meeting: 1954, to be announced.

Fifth Inter-American Congress of Radiology
General Secretary, Dr. Eugene P. Pendergrass, 3400 Spruce St., Philadelphia 4, Pa. Meeting at Shoreham Hotel, Washington, D. C., April 24–30, 1955.

Alabama Radiological Society
Secretary, Dr. J. A. Meadows, Jr., Medical Arts Bldg., Birmingham 5, Ala. Meets time and place Alabama State Medical Association.

Arizona Radiological Society
Secretary, Dr. R. Lee Foster, 15 E. Monroe, Phoenix, Ariz. Two regular meetings a year. The annual meeting at time and place of State Medical Association and interim meeting six months later.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Atlanta Radiological Society
Secretary, Dr. Albert A. Rayle, Jr., 478 Peachtree St., N.E., Atlanta, Ga. Meets monthly, except during three summer months, on second Friday evening.

Brooklyn Roentgen Ray Society
Secretary, Dr. J. J. Daversa, 345 75th St., Brooklyn, N. Y. Meets monthly fourth Tuesday, October through April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. F. A. Riebel, 15 West Goodale St., Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Columbus Athletic Club, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. Elbert K. Lewis, 6337 S. Harvard Ave., Chicago, Ill. Meets second Thursday of each month, October to April inclusive at the University Club.

Cleveland Radiological Society
Secretary, Dr. Mortimer Lubert, 10515 Carnegie Ave., Cleveland 6, Ohio. Meetings at 6:45 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
President, Dr. W. P. Stampfli, St. Luke's Hospital, Denver, Colo. Meets third Friday of each month at University of Colorado Medical Center or at Denver Athletic Club.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday October and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. J. C. Cook, Harper Hospital, Detroit, Mich. Meets monthly first Thursday, October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434 30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. A. Judson Graves, 2002 Park St., Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert M. Tankesley, 218 Doctors Bldg., Atlanta, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Greater Miami Radiological Society
Secretary, Dr. E. H. Bryson, 273 Alhambra Circle, Coral Gables, Fla. Meets third Wednesday of each month at Veterans Administration Building.

Houston Radiological Society
Secretary, Dr. Harry Fishbein, 324 Medical Arts Bldg., Houston 2, Texas. Meets fourth Monday each month at Hermann Hospital.

Idaho State Radiological Society
Secretary, Dr. Alfred M. Stone, St. Lukes Hospital, Boise, Idaho. Meets annually at same time as Idaho State Medical Association.

Illinois Radiological Society
Secretary, Dr. Stephen L. Casper, 1101 Maine St., Quincy, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa Radiological Society
Secretary, Dr. James T. McMillan, 1104 Bankers Trust Bldg., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society
Secretary, Dr. Willis L. Beeler, M.D., 700 Kansas Ave., Topeka, Kansas. Meets in spring with State Medical Society, and in winter on call.

Kentucky Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Seelbach Hotel, Louisville.

Kings County Radiological Society
Secretary, Dr. Marcus Wiener, 1430 48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. George Jacobson, 1200 N. State St., Los Angeles 33, Calif. Meets second Wednesday each month at Los Angeles County Medical Association Building, Los Angeles.

* Secretaries of societies are requested to send timely information promptly to the Editor.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Maine Radiological Society
Secretary, Dr. Hugh Allan Smith, Eastern Maine General Hospital, Bangor, Maine. Meets in early spring, June and fall.

Miami Valley Radiological Society
Secretary, Dr. G. A. Nicoll, 134 Apple St., Dayton, Ohio. Meets second Friday of fall and winter months.

Mid-Hudson Radiological Society
Secretary, Dr. J. W. Walton, Horton Memorial Hospital, Middletown, N. Y. Meets 8:30 P.M., fourth Tuesday each month, September to May.

Milwaukee Roentgen Ray Society
Secretary, Dr. T. J. Pfeffer, 839 N. Marshall St., Milwaukee 2, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. John R. Hodgson, Mayo Clinic, Rochester, Minn. Meets three times annually, once at time of State Med. Assn., and in spring and fall.

Mississippi Radiological Society
Secretary, Dr. John W. Evans, 117 N. President St., Jackson, Miss. Meets third Thursday of each month at the Rotisserie Restaurant at 6:30 P.M.

Montana Radiological Society
Secretary, Dr. Grant P. Raitt, Suite 413 Medical Arts Bldg., Billings, Montana. Will meet at least once a year.

Nassau Radiological Society
Secretary, Dr. Joseph J. La Vine, 259 North Grand Ave., Baldwin, N. Y. Meets second Tuesday of the month in February, April, June, October and December.

Nebraska Radiological Society
Secretary, Dr. J. F. Kelly, Jr., 816 Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Stanley M. Wyman, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Harvard Club.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. J. R. Freid, 1049 Park Ave., New York 28, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 1420 E. Fifth St., Charlotte 4, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings by announcement.

Northeastern New York Radiological Society
Secretary, Dr. John F. Roach, Albany Hospital, Albany, N. Y. Meets in Albany area on second Wednesdays of October, November, March and April. Annual meeting will be held in May or June, 1953.

Northern California Radiological Society
Secretary, Dr. H. B. Stewart, 2920 Capitol Ave., Sacramento, Calif. Meets at dinner last Monday of each month, September to June.

Ohio State Radiological Society
Secretary, Dr. Willis S. Peck, 1838 Parkwood Ave., Toledo 2, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. John R. Danstrom, Medical Arts Bldg., Oklahoma City, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. J. Richard Raines, 214 Medical-Dental Bldg., Portland 5, Oregon. Meets monthly from October to June on the second Wednesday of each month at 8:00 P.M. at the University Club.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. H. M. Stauffer, Temple University Hospital, Philadelphia 40, Pa. Meets first Thursday of each month, at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Donald H. Rice, Western Pennsylvania Hospital, Pittsburgh 24, Pa. Meets 6:00 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Radiological Section, Baltimore Medical Society
Secretary, Dr. H. Leonard Warres, 2337 Eutaw Place, Baltimore 17, Md. Meets third Tuesday each month, September to May, inclusive.

Radiological Section, Connecticut Medical Society
Secretary, Dr. William A. Goodrich, 85 Jefferson St., Hartford 6, Conn. Meetings are held bi-monthly.

Radiological Section, Medical Society of the District of Columbia
Secretary, Dr. U. V. Wilcox, 915 Nineteenth St., N. W., Washington 6, D. C. Meets Medical Society Library, third Thursday of January, March, May and October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr Roy G. Giles, Temple, Texas.

Radiological Society of Greater Cincinnati
Secretary, Dr. Harry K. Hines, 2508 Auburn Ave., Cincinnati 19, Ohio. Meets monthly from September to June on first Monday of each month at 7:30 P.M. at the Cincinnati General Hospital.

Radiological Society of Hawaii
Secretary, Colonel Alexander O. Haff, M.C., Tripler Army Hospital, Honolulu, T. H. Meets third Friday of each month at Tripler Army Hospital.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Radiological Society of New Jersey
Secretary, Dr. Salomon Silvera, 921 Bergen Ave., Jersey City, N. J. Meets at Atlantic City at time of State Medical Society and in November in Newark, N. J.

Richmond County Radiological Society
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. A. Gordon Ide, 277 Alexander St., Rochester 7, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Next annual meeting, Shirley-Savoy Hotel, Denver, Colo., August 20, 21 and 22, 1953.

St. Louis Society of Radiologists
Secretary, Dr. Francis O. Trotter, Jr., 634 N. Grand Blvd., St. Louis 3, Mo. Meets fourth Wednesday each month, except June, July, August and September.

San Antonio-Military Radiological Society
Secretary, Dr. Hugo F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Texas. Meets Brook Army Medical Center Officers Mess first Monday of each month.

San Diego Radiological Society
Secretary, Dr. C. W. Bruner, 2456 Fourth Ave., San Diego 1, Calif. Meets first Wednesday of each month at the University Club.

San Francisco Radiological Society
Secretary, Dr. I. J. Miller, 2680 Ocean Avenue, San Francisco 27, Calif. Meets quarterly.

Section on Radiology. California Medical Association
Secretary, Dr. Calvin L. Stewart, 2330 First Ave., San Diego 1, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. George E. Irwin, Jr., 703 N. East St., Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Bay Radiological Society
Secretary, Dr. W. H. Graham, 634 East Santa Clara St., San Jose 12, Calif. Meets second Wednesday each month.

South Carolina Radiological Society
Secretary, Dr. William A. Klauber, Self Memorial Hospital, Greenwood, S. C. One meeting with South Carolina Medical Association in May, others arranged by President.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, Newell Hospital, Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort worth 4, Texas. Next meeting, Dallas, Texas, Jan. 29 and 30, 1954.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May, Service Memorial Institute.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. John N. Burkey, 555 Medical-Dental Bldg., Seattle 1, Wash. Meets fourth Monday of each month from September through May.

Westchester Radiological Society
Secretary, Dr. Clifford C. Baker, Harwood Bldg., Scarsdale, N. Y. Meets on third Tuesdays of January and October and on two other dates.

Wisconsin Radiological Society
Secretary, Dr. Irving I. Cowan, 425 E. Wisconsin Ave., Milwaukee 2, Wis. Special meetings held during year; annual meeting each spring at Madison, Wis.

X-Ray Study Club of San Francisco
Secretary, Dr. Wm. W. Saunders, Veterans Administration Hospital, San Francisco 21, Calif. Meets monthly, third Thursday, at 7:45 P.M., Lane Hall, Stanford University Hospital.

Cuba, Mexico, Puerto Rico and Central America

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

Asociación Puertorriqueña de Radiología
Secretary, Dr. Rafael A. Blanes, Ave. Ponce de Leon 1175, Santurce, P. R.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sanchez, Apartado No. 86, Panama, R. de P. Meets monthly in a department of radiology of a local hospital, chosen at preceding meeting.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary-Treasurer, Dr. D. L. McRae, Suite 305, 1555 Summerhill Avenue, Montreal 25, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Française d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East Montreal, P. Q.

College of Radiologists (Australia and New Zealand)
Honorary Secretary, Dr. Alan R. Colwell, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W.

South America

Asociacion Argentina de Radiologia
Secretary, Dr. Lidio G. Mocsa, Avda. Gral. Paz 151, Córdoba, Argentina. Meetings held monthly.

Sociedad Argentina de Radiologia, Junta Central, Buenos Aires
Secretary, Dr. Oscar F. Noguera, Santa Fe 1171, Buenos Aires. Meetings are held monthly.

Sociedad Argentina de Radiologia, Filial del Litoral, Sede, Rosario
President, Dr. Francisco P. Cifarelli. Meets monthly second Wednesday, 19 ha 663 Italia St., Rosario, Santa Fe.

Sociedad Argentina de Radiologia, Filial del Sur, Sede, La Plata
President, Prof. Dr. Jose P. Uslenghi, La Plata. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Centro Sede, Cordoba
President, Prof. Dr. David L. Caro, Corro 86, Cordoba. Meets monthly.

Sociedad Argentina de Radiologia, Filial del Norte, Sede, Tucuman
President, Dr. Julio Mendez Valladares, Rivadavia 358, Tucuman. Meets monthly.

Sociedad Argentina de Radiologia, Filial de Cuyo Sede, Mendoza.
President, Dr. J. A. Orfila, Hospital Central, Mendoza. Meets monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644 São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Colombiana de Radiologia
Secretary, Dr. Luis E. Lozano B., Carrera 12 No. 20-69, 6° piso, Bogota, Colombia. Meets last Thursday of each month.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado 2306, Lima, Peru. Meets monthly except during January,

February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini, Av. Agraciada 1464, Piso 13, Montevideo, Uruguay.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Allies, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Société Française d'Electro-Radiologie Médicale, and its branches: Société du Sud-Ouest, du Littoral Méditerranéen, du Centre et du Lyonnais, de l'Ouest, de l'Est, et d'Alger et d'Afrique du Nord
Secretary-General, Dr. A. Dariaux, 9 bis, Boulevard Rochechouart, Paris IX^{e}, France.

Ceskoslovensk společnost pro rëntgenologii a Radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10:30, X-Ray Dept., Akademia Gdansk.

Wrocław Section Polish Society of Radiology
Secretary, Dr. R. Marciniak, Akad. Medyczna Wrocław, 68 Curie-Skłodowska St., Wrocław, Poland. Meets first Sunday of each month, X-Ray Department, Akademia Medyczna Wrocław.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
Secretary, Dr. Max Hopf, Effingerstrasse 47, Bern, Switzerland.

Deutsche Roentgen-Gesellschaft
Secretary, Dr. Peter Kröker, (22a) Essen-Helsingen, Westpreussenstrasse 21. Meets annually in different towns.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano. Torino, Italy. Meets biannually.

India

Indian Radiological Association
Secretary, Dr. R. F. Sethna, Navsari Building, Hornby Road, Bombay 1, India.

PRELIMINARY PROGRAM

FIFTY-FOURTH ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

THE Fifty-fourth Annual Meeting of the American Roentgen Ray Society will be held at the Netherland Plaza Hotel, Cincinnati, Ohio, September 29 to October 2, 1953.

The Executive Council will meet at noon on Sunday, September 27.

The annual Golf Tournament for the Willis F. Manges trophy will be held on Monday, September 28, at the Kenwood Country Club. The annual Golf Dinner will be held at the same place after the tournament.

Dr. Paul Chesley Hodges of Chicago, Illinois, will give the Caldwell Lecture on Tuesday evening, September 29. The Annual Banquet will be held on Thursday evening, October 1.

The Instruction Courses will begin at 3:00 P.M. on Tuesday, Wednesday and Thursday, and at 1:30 P.M. on Friday. Elsewhere in this issue will be found the detailed plan of the courses.

The Scientific Program, which has been arranged by President-elect Harry M. Weber and his Program Committee, is as follows:

Tuesday, September 29, 1953

8:30 A.M. Call to order, Fifty-fourth Annual Meeting, Charles L. Martin, M.D., President.

Address of Welcome: Stanley E. Dorst, M.D., Dean of the College of Medicine of the University of Cincinnati.

Installation of the President-Elect, Harry M. Weber, M.D., Rochester, Minnesota, by President Martin and the Chairman of the Executive Council, Clyde A. Stevenson, M.D., Temple, Texas.

Inaugural Address: President Harry M. Weber, M.D.

9:00 A.M.

Paper No.

1. Radiology and the National Research Council. Eugene P. Pendergrass, M.D., Philadelphia, Pa.
2. The Roentgenologic Diagnosis of Aneurysm of the Thoracic Aorta, with Particular Reference to Study in the Right Posterior Oblique Position. George Levene, M.D., Edward N. Burke, M.D. (by invitation), and Dolores C. Arnois, M.D. (by invitation), Boston, Mass. Discussion by John W. Kirklin, M.D. (by invitation), Rochester, Minn.
3. Angiocardiography in Children. Sven Roland Kjellberg, M.D. (by invitation), Stockholm, Sweden.
4. Lobar Emphysema in Children. E. F. Van Epps, M.D. (by invitation), and Dale H. Davies, M.D. (by invitation), Iowa City, Iowa. Discussion of papers by Dr. Kjellberg, and Drs. Van Epps and Davies by E. B. D. Neuhauser, M.D., Boston, Mass.
5. Roentgenographic Demonstration of Avulsion of Nerve Roots. J. E. Whiteleather, M.D., Memphis, Tenn. Discussion by Harold O. Peterson, M.D., St. Paul, Minn.

Symposium: A Practical Consideration of Mitral Stenosis. Moderator: D. G. Pugh, M.D., Rochester, Minn.

6. Pathologic Aspects: Jesse E. Edwards, M.D. (by invitation), Rochester, Minn.
7. Clinical Aspects and Physiologic Changes: Robert G. Tompkins, M. D. (by invitation), Rochester, Minn.
8. Surgical Observations: John W. Kirklin, M.D. (by invitation), Rochester, Minn.
9. The Roentgenologic Contribution: D. G. Pugh, M.D. Discussion by Leo G. Rigler, M.D., Minneapolis, Minn.

12 M. Luncheon and Study of Scientific and Technical Exhibitis.

12:30 P.M. Fourth Annual Past Presidents Luncheon.

3:00 P.M. Instruction Courses.

4:30 P.M. Executive Business Session of the Society.

Tuesday Evening, September 29, 1953

Eight-Thirty o'Clock
The Caldwell Lecture
"Normal Bone, Diseased Bone, Dead Bone"
by
Paul C. Hodges, M.D., Ph.D.
Professor and Head of the Department of Radiology, University of Chicago, Chicago, Illinois
Introduction—Russell H. Morgan, M.D., Baltimore, Maryland
Presentation of Medal to Dr. Hodges—President Weber

Wednesday, September 30, 1953

9:00 A.M.

10. Isodose Curves for Intracavity Therapy. E. Dale Trout, B.S., Sc.D. (by invitation), John P. Kelley, B.S., (by invitation), and Arthur C. Lucas (by invitation), Milwaukee, Wis. Discussion by Ralph M. Caulk, M.D., Washington, D. C.
11. A New Phototimer for Roentgenography. Eugene L. Saenger, M.D., and Philip Ohmart (by invitation), Cincinnati, Ohio. Discussion by Paul C. Hodges, M.D., Chicago, Ill.
12. Remote Fluoroscopic Control of Radiation Therapy. Russell H. Morgan, M.D., Ralph E. Sturm, Physicist (by invitation), Lowell S. Miller, M.D. (by invitation), and Daniel J. Torrance (by invitation), Baltimore, Md. Discussion by Paul C. Hodges, M.D., Chicago, Ill.

Symposium: Radiation Protection. Moderator: Andrew H. Dowdy, M.D., Los Angeles, Calif.

13. Introduction. Fundamental Aspects of the Problem. Andrew H. Dowdy, M.D., Los Angeles, Calif.
14. Instruments for Measuring Exposure of Personnel. Herbert M. Parker, M.Sc., F. Inst. P. (by invitation), Richland, Wash.
15. Exposure Problems in Diagnostic Roentgenology. George C. Henny, M.D., Philadelphia, Pa.
16. Exposure Problems in Therapeutic Radiology. Harold O. Wyckoff (by invitation), National Bureau of Standards, Washington, D. C.
17. Health Problems in a Radioisotope Laboratory. John Hale, A.B. (by invitation), Philadelphia, Pa. Discussion by L. D. Marinelli, Ph.D. (by invitation), Chicago, Ill.

12 M. Luncheon and Study of Scientific and Technical Exhibits.

3:00 P.M. Instruction Courses.

Thursday, October 1, 1953

9:00 A.M.

18. Roentgen Studies of the Effects on the Small Intestine by Emotional Disturbances. Jack Friedman, M.D. (by invitation), Minneapolis, Minn.
19. Roentgen Studies of the Small Intestine in Sprue. Richard H. Marshak, M.D., Bernard S. Wolf, M.D., and David Adlersberg, M.D. (by invitation), New York, N. Y.
20. Amyloidosis of the Small Intestine. Ross Golden, M.D., New York, N. Y. Discussion of papers by Dr. Friedman, Dr. Marshak, *et al.*, and Dr. Golden by Leon Schiff, M.D. (by invitation), Cincinnati, Ohio, and F. J. Hodges, M.D., Ann Arbor, Mich.
21. Venographic Clues as to Localization of Intracranial Masses. P. A. Riemenschneider, M.D. (by invitation), and A. D. Ecker, M.D. (by invitation), Syracuse, N. Y. Discussion by Philip J. Hodes, M.D., Philadelphia, Pa.
22. Mobile Explosion- and Shock-proof Roentgenographic Equipment in the Operating Room for Cholangiography and Hip Nailing. Barton R. Young, M.D., and Robert L. Scanlan, M.D., Philadelphia, Pa.
23. Renal Arteriography. Arthur T. Evans, M.D. (by invitation), Cincinnati, Ohio.
24. Abdominal Aortography for the Roentgen Demonstration of the Liver and Spleen. Leo G. Rigler, M.D., and Paul C. Olfelt, M.D. (by invitation), Minneapolis, Minn.
25. Abdominal Aortography in Intrinsic Disease of the Abdominal Aorta and Its Larger Branches. Benjamin Felson, M.D. (by invitation), Cincinnati, Ohio. Discussion of papers of Dr. Evans, Drs. Rigler and Olfelt, and Dr. Felson by Paul C. Swenson, M.D., Philadel-

phia, Pa., and Fred O. Coe, M.D., Washington, D. C.

12 M. Luncheon and Study of Scientific and Technical Exhibits.

3:00 P.M. Instruction Courses.

4:30 P.M. Executive Business Session of the Society.

Thursday Evening, October 1, 1953

Seven o'Clock
Cocktails—Pavillon Caprice
Eight o'Clock
Annual Banquet—Pavillon Caprice
Presentation of Scientific Awards

Friday, October 2, 1953

9:00 A.M.

26. Necrosis of the Brain Following Roentgen Irradiation. G. S. Dugger, M.D. (by invitation), J. G. Stratford, M.D. (by invitation), and Jean Bouchard, M.D. (by invitation), Montreal, Quebec, Canada. Discussion by Frank H. Mayfield, M.D. (by invitation), Cincinnati, Ohio.

27. Medical Aspects of High Energy Electron Beams. Lewis L. Haas, M.D. (by invitation), Roger A. Harvey, M.D., John L. Laughlin, Ph.D. (by invitation), John W. Beattie, B.S. (by invitation), and Walter J. Henderson, B.S. (by invitation), Chicago, Ill. Discussion by Hugh F. Hare, M.D., Los Angeles, Calif.

28. Quantitative Studies of the Time-Dose (Recovery) Factor Based on Recurrent Carcinoma of the Breast. Milton Friedman, M.D., and Alexander W. Pearlman, M.D. (by invitation), New York, N. Y. Discussion by Charles L. Martin, M.D., Dallas, Texas.

Symposium: Treatment of Cancer of the Oral Cavity. Moderator: J. A. del Regato, M.D., Colorado Springs, Colorado.

29. Introduction. General Statement of the Problem. J. A. del Regato, M.D.

30. Surgical Treatment of Cancer of the Tongue and Floor of the Mouth. Danely P. Slaughter, M.D. (by invitation), Chicago, Ill.

31. Radiation Therapy of Cancer of the Tongue and Floor of the Mouth. Clifford L. Ash, M.D. (by invitation), Toronto, Ontario, Canada.

32. Surgical Treatment of Cancer of the Buccal Mucosa and Lower Gingiva. John J. Medlin, M.D. (by invitation), Columbia, Missouri.

33. Radiation Therapy of Cancer of the Buccal Mucosa and Lower Gingiva. Isadore Lampe, M.D., Ph.D., Ann Arbor, Mich.

12 M. Luncheon and Study of Scientific and Technical Exhibitis.

1:30 P.M. Instruction Courses.

AMERICAN ROENTGEN RAY SOCIETY

SECTION ON INSTRUCTION

B. R. Kirklin, M.D., *Director*

Titles and Abstracts of Courses Offered

Fifty-fourth Annual Meeting
Netherland Plaza Hotel, Cincinnati, Ohio

September 29–October 2, 1953

PRESIDENT-ELECT WEBER, with the approval of the Executive Council, has requested that the Section on Instruction be continued for the annual meeting of 1953. His program has been arranged so that the Instruction Courses will be offered between the hours of 3:00 P.M. and 4:30 P.M. on Tuesday, Wednesday, and Thursday, and 1:30 and 3:00 P.M. on Friday. One course will be offered this year in each of the eleven rooms assigned for this purpose. Thus a total of 90 minutes has been made available for each course. This makes it possible for instructors to devote sufficient time to discussion and to conference with those attending the courses. No other official activity has been scheduled for these times.

The Section of Instruction presents for 1953:

(1) Two Panel Discussions on the uses of radioactive isotopes:

(a) Course 101, relating to the use of the isotopes in diagnosis,

(b) Course 102, relating to the use of the isotopes in therapy.

(2) A Special Course given under the auspices of the American College of Radiology (Course 201).

(3) A Special Course on the construction and operation of a library of teaching roentgenograms (Course 202).

(4) Three courses (Courses 301, 302, 303), dealing largely with fundamental physical and biological aspects of Radiology, with a faculty of three instructors and covering three periods.

(5) Two Sequential Courses in Diagnostic Roentgenology (Courses 401A, 401B, 402A, 402B), and one in Therapeutic Radiology (Course 508A, 508B), with a faculty of five instructors and covering six periods.

(6) Seven single period courses in Therapeutic Radiology (Courses 501–507) with a faculty of seven instructors and covering seven periods.

(7) Twenty-two single period courses (Courses 601–622) in Diagnostic Roentgenology with a faculty of nineteen instructors and covering twenty-two periods.

GENERAL INFORMATION

THE FACULTY

Paul C. Aebersold, Ph.D., Chief of the Isotope Branch, U. S. Atomic Energy Commission, Oak Ridge, Tennessee.

W. E. Baensch, M.D., Professor of Radiology, Georgetown University School of Medicine, Washington, D. C., Head of the Departments of Radiology at Georgetown University Hospital, Washington, D. C., and Arlington Hospital, Arlington, Virginia; Senior Consultant, Veterans Administration, Washington, D. C.

W. Edward Chamberlain, M.D., Professor of Radiology, Temple University Medical School and Head of the Department of Radiology, Temple University Hospital, Philadelphia, Pennsylvania.

Fred O. Coe, B.S., M.D., Radiological Clinic of Drs. Groover, Christie and Merritt, Radiologist to Doctors Hospital, Washington, D. C.

Robert N. Cooley, M.D., Associate Professor, Department of Radiology, Johns Hopkins Hospital, Baltimore, Maryland.

Kenneth E. Corrigan, Ph.D., Director, Research Division, Harper Hospital, Detroit, Michigan.

Charles T. Dotter, M.D., Professor of Radiology, University of Oregon Medical School, Portland, Oregon.

H. B. Elkins, M.D., Associate Professor of Radiology, University Hospitals, Iowa City, Iowa.

Edwin C. Ernst, M.D., Director, Radiological Department, Barnard Free Skin and Cancer Hospital, St. Louis, Missouri.

Arthur T. Evans, M.D., Attending Urologist, Christ Hospital, Children's Hospital, Cincinnati General Hospital; Instructor in Urology, University of Cincinnati, Cincinnati, Ohio.

Titus C. Evans, Ph.D., Head, Radiation Research Laboratory, College of Medicine, State University of Iowa, Iowa City, Iowa.

Jack Friedman, M.D., Radiologist, University of Minnesota, and Mt. Sinai Hospital, Minneapolis, Minnesota.

Joseph H. Gast, Sc.D., Professor and Chairman of the Department of Biochemistry, Baylor University College of Medicine; Chairman, Methodist Hospital-Baylor Isotope Committee, Houston, Texas.

C. Allen Good, M.D., M.S., Associate Professor of Radiology, Mayo Foundation, University of Minnesota; Consultant, Section of Diagnostic Roentgenology, Mayo Clinic, Rochester, Minnesota.

Hugh F. Hare, M.D., Radiologist, Los Angeles Tumor Institute, Los Angeles, California.

Fred J. Hodges, M.D., Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

John F. Holt, M.D., Associate Professor of Roentgenology, University of Michigan, Ann Arbor, Michigan.

Sven Roland Kjellberg, M.D., Karolinska Sjukhuset, Stockholm, Sweden.

W. C. MacCarty, Jr., M.D., Assistant Professor of Radiology, Dartmouth Medical School and Member, Department of Radiology, Hitchcock Clinic and Mary Hitchcock Memorial Hospital, Hanover, New Hampshire.

A. S. Macmillan, M.D., Radiologist, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

W. L. McLaughlin, M.D., Assistant Professor of Urology, Dartmouth Medical School, and Member, Department of Urology, Hitchcock Clinic and Mary Hitchcock Memorial Hospital, Hanover, New Hampshire.

L. D. Marinelli, Ph.D., Argonne National Laboratory, Lemont, Illinois.

Richard H. Marshak, M.D., Associate Radiologist, Mt. Sinai Hospital, New York, New York.

Charles L. Martin, M.D., Professor of Radiology, Southwestern Medical College, Dallas Texas.

Isadore Meschan, M.D., Professor and Head, Department of Radiology, University of Arkansas School of Medicine, Little Rock, Arkansas.

Earl R. Miller, M.D., Professor of Radiology, University of California, Berkeley, California.

Russell H. Morgan, M.D., Professor, Department of Radiology, Johns Hopkins Hospital, Baltimore, Maryland.

Edward B. D. Neuhauser, M.D., Associate in Radiology, Harvard Medical School, Boston, Massachusetts; Radiologist to The Infant's and to The Children's Hospitals of Boston, Boston, Massachusetts.

H. M. Parker, M.Sc., F. Inst. P., Nucleonics Division, General Electric Company, Richland, Washington.

H. F. Plaut, M. D., Assistant Chief of Radiology, Veterans Administration Center, Dayton, Ohio.

Harold O. Peterson, M.D., Clinical Associate, Professor of Radiology, University of Minnesota, Minneapolis, Minnesota; Radiologist to Miller Hospital and Children's Hospital, St. Paul, Minnesota.

David G. Pugh, M.D., Assistant Professor of Radiology, Mayo Foundation, University of Minnesota; Consultant, Section of Diagnostic Roentgenology, Mayo Clinic, Rochester, Minnesota.

Juan A. del Regato, M.D., Assistant Professor of Radiology, University of Colorado Medical School; Director, Penrose Cancer Hospital, Colorado Springs, Colorado.

Robert Robbins, M.D., Associate Professor of Radiology, Chief of the Division of Therapy, Department of Radiology, Temple University, Philadelphia, Pennsylvania.

Maurice D. Sachs, M.D., Chief, Radiology Service, Veterans Administration Hospital, Cleveland, Ohio.

Robert L. Scanlan, M.D., Radiologist, Germantown Hospital, and Instructor in Radiology, Temple University, Philadelphia, Pennsylvania.

John G. Trump, Sc.D., Associate Professor, Massachusetts Institute of Technology, Cambridge, Massachusetts.

CONFERENCE PERIODS

Tuesday, Wednesday, Thursday and Friday Afternoons

Single Periods.....3:00–4:30 P.M.
(Tuesday, Wednesday and Thursday)

Single Periods.....1:30–3:00 P.M.
(Friday)

Location

All courses will be given in Parlors A, B, C, D, E, F, G, H, I, J, and L, located on the Fourth Floor of the Netherland Plaza Hotel. Full information will be obtainable at the registration desk of the Section on Instruction which will be located nearby.

Attendants will be at this desk from 1:00 P.M. to 5:00 P.M. on Sunday, September 27, and on the remaining days of the meeting from 8:30 A.M. until 4:30 P.M.

Code

The instruction periods will be designated with the following code:

T, Tuesday.....3:00–4:30 P.M.
W, Wednesday.....3:00–4:30 P.M.
Th, Thursday.....3:00–4:30 P.M.
F, Friday.....1:30–3:00 P.M.

How to Register and Obtain Tickets for the Instruction Courses

Admission to the Instruction Courses will be by ticket only.

Following the list of titles and abstracts is a *general order sheet.* First, second, and third choices for each period should be selected carefully and indicated on the order sheet. The rooms in which the courses will be given are moderately large ones, but it will be necessary to place a limitation on the number of persons who will be able to attend individual courses. It is estimated that the number who can attend will range between 50 and 100 persons. If the directions given on the order sheet are followed explicitly, errors in completing reservations for the courses will be minimized.

It is possible for one to attend only four periods of instruction, so the condensed schedule given on the last pages of this program should be consulted with care when registering and ordering tickets for the courses.

Reservations for the courses will be made in the order in which the order forms are received. Those who are not members of the American Roentgen Ray Society will be charged the nominal fee of $1.50 per period of instruction, or a maximum fee of $5.00 for four periods. Full-time graduate students in radiology will be admitted without fee, but they will be required to make application for the courses they wish to attend.

Previous to September 12, 1953, the order forms should be sent to the Director, Dr. B. R. Kirklin, Mayo Clinic, Rochester, Minnesota. After September 12, 1953, the order forms should be sent directly to Dr. B. R. Kirklin, Netherland Plaza Hotel, Cincinnati, Ohio.

To avoid the confusion always existing on the first days of the meeting, a set of tickets will be mailed to each person whose registration for courses reaches the office of the Director on or before September 12, 1953. The envelope containing the tickets will be labelled in a conspicuous manner. Registrants are urged to be on the lookout for their tickets, and to bring them to the meeting. The success of this plan depends entirely on the cooperation of the registrants.

In case the courses are not filled by the time of the meeting, tickets will be available at the registration desk of the Section on Instruction, on Sunday, September 27, and thereafter during the meeting.

Holders of Tickets

Those who do not have the proper ticket for the course they wish to attend will not be permitted to enter the room. An attendant will be at the door of each conference room to collect the tickets.

DESCRIPTION OF COURSES

COURSE 101

Room I **Period: T**

PANEL

KENNETH E. CORRIGAN, Ph.D., Moderator
Detroit, Michigan

E. R. Miller, M.D.	**H. B. Elkins, M.D.**
Robert Robbins, M.D.	**Titus C. Evans, Ph.D.**
Joseph H. Gast, Sc.D.	**L. D. Marinelli, Ph.D.**

A Round Table Workshop on the Use of Radioisotopes in Diagnosis

Radiologists using or planning to use radioisotopes in diagnosis might like to exchange ideas and information about equipment and techniques. In this session the panel will attempt to give the best answers to questions and help in the solution of problems, with all attendants free to participate in either questions or answers. *Those interested are invited to send questions and present problems to the moderator or to individual members of the panel in advance of the meeting.*

COURSE 102

Room I **Period: W**

PANEL

KENNETH E. CORRIGAN, Ph.D., Moderator
Detroit, Michigan

E. R. Miller, M.D.	**H. B. Elkins, M.D.**
Robert Robbins, M.D.	**Titus C. Evans, Ph.D.**
Paul C. Aebersold, Ph.D.	**H. M. Parker, M.Sc.**

A Round Table Workshop on the Use of Radioisotopes in Therapy

Radiologists using or planning to use radioisotopes in therapy might like to exchange ideas and information about equipment and techniques. In this session the panel will attempt to give the best answers to questions and help in the solution of problems, with all attendants free to participate in either questions or answers. *Those interested are invited to send questions and present problems to the moderator or to individual members of the panel in advance of the meeting*

COURSE 201

Room I **Period: F**

W. EDWARD CHAMBERLAIN, M.D.
Philadelphia, Pennsylvania

Radiologic Ethics, Administration and Economics

(This course is given under the auspices of The American College of Radiology)

This period will be devoted to a frank discussion of ethical, administrative and economic problems which the radiologist must face in developing and maintaining a satisfactory arrangement to practice radiology in a hospital. Examples of desirable arrangements will be presented. Pitfalls that lead to unsatisfactory relationships will be discussed. It will be emphasized that the radiologist must from the very beginning establish himself in the status of a consultant. The important aspects of developing and maintaining the proper ethical and economic relationships between the patient and the radiologist will be presented in a frank and thorough manner.

COURSE 202

Room F **Period: T**

FRED J. HODGES, M.D.
Ann Arbor, Michigan

A Library of Teaching Films (Its Construction and Operation)

The need for readily accessible demonstration films of normal and abnormal subjects dealing with all aspects of roentgen diagnosis confronts almost every radiologist. In his daily contacts with medical consultants in other fields, the radiologist has practical need of such material readily available to substantiate his reasons for opinions rendered on current case material. His usefulness in extending radiologic instruction to interns and house officers is greatly increased by the possession of a well balanced and logically catalogued library of teaching films. For those radiologists who are heavily engaged in undergraduate medical instruction as well as at the graduate and post-graduate levels, carefully selected teaching material in film form is an essential part of his teaching equipment.

A method, which has been used at the University of Michigan since 1935, which is geared to the filing and cross-indexing system for roentgen-ray records adopted in that year, will be explained and illustrated. The great advantage of continuous additions and replacements in the library file will be emphasized. Details of the method employed will be clarified. The manner in which the basic library of teaching films can be linked with projection material in the form of lantern slides or strip film will be illustrated.

Experience has shown that the most successful

plan for the acquisition of teaching material is to make it a part of each day's routine of current film interpretation rather than to attempt the building of a teaching library at one sitting. Suggestions will be offered regarding the optimum size of teaching files depending upon the anticipated use of such material.

This refresher material will be amplified by a corresponding demonstration in the scientific exhibits.

COURSE 301

Room A **Period: F**

TITUS C. EVANS, Ph.D.
Iowa City, Iowa

Fundamental Aspects of Effects of Ionizing Radiation

The differences between the interaction of material with ionizing and with non-ionizing radiation will be discussed briefly.

Our present concept of the mechanism whereby the energy in a roentgen-ray beam is transferred to tissue so as to produce first physical, then chemical, and finally biologic changes will be discussed.

Mention will be made of the various theories of biologic action which attempts to stress the relative importance of the various processes whereby the radiation energy has been absorbed.

There will be a brief discussion of variation in and modification of radiosensitivity. The factors of radiation intensity and radiation wavelengths will be discussed as regards their biologic effectiveness.

COURSE 302

Room A **Period: Th**

H. M. PARKER, M.Sc., F.Inst.P.
Richland, Washington

Radiation Dosimetry

1. Roentgen rays and gamma rays:
   Advantages of an ionization method.
   Interpretation of the original definition of the "roentgen."
   Energy absorption and the Bragg-Gray Principle.
   Interpretation of the current definition of the "roentgen."
   The gamma-roentgen.
   Complexities with multimillion volt radiation.
2. Other radiations:
   Extension of the roentgen to particulate radiations.
   Gray's Energy Unit.
   The "rep."
   Applications to radioisotope therapy and neutron therapy.
   Other proposals for physical dose units.
3. Factors of biological effectiveness:
   Relative biological effectiveness.
   The "rem."
   Scale of relation between "rep" and "rem."
   Severe limitations of the "rem."

COURSE 303

Room A **Period: W**

RUSSELL H. MORGAN, M.D.
Baltimore, Maryland

The Place of Electronics in Roentgenology

The rapid development of the field of electronics has reflected itself in an ever increasing application of electronic methods to the science of radiology. The basic principles which are involved in many of these electronic applications will be discussed in detail. The course will include material on a variety of practical electronic instruments that are finding an increasing use in radiology. These include scintillation counters, Geiger-Müller counters, the electronic components of the various screen intensification systems and the electronic components of such diagnostic apparatus as automatic phototimers and electronic timers.

COURSE 401A

Room E **Period: T**

W. C. MacCARTY, JR., M.D.
W. L. McLAUGHLIN, M.D.
Hanover, New Hampshire

Excretory Urography

PART I

I. History of Excretory Urography.
II. Technique.
   Preparation of the patient.
   Roentgenographic technique.
   Selection of urographic media.
III. Congenital Anomalies.
IV. Extraurinary Lesions Affecting the Urinary Tract.

COURSE 401B

Room E **Period: W**

W. C. MacCARTY, JR., M.D.
W. L. McLAUGHLIN, M.D.
Hanover, New Hampshire

Excretory Urography

PART II

V. Acquired Lesions of the Urinary Tract.
   Physiological conditions.
   Inflammatory disease.
   Stones.
   Prostatic hypertrophy.
   Traumatic conditions.

VI. Neoplasms.
Benign.
Malignant.

COURSE 402A

Room B **Period: Th**

CHARLES T. DOTTER, M.D.
Portland, Oregon

Angiocardiography

PART I

The conduct of the angiocardiographic examination.
Contrast media.
The normal angiocardiogram.

COURSE 402B

Room B **Period: F**

CHARLES T. DOTTER, M.D.
Portland, Oregon

Angiocardiography

PART II

The abnormal angiocardiogram.
Attendants are urged to bring interesting or difficult angiocardiograms to the session for open discussion.

COURSE 501

Room B **Period: T**

E. C. ERNST, M.D.
St. Louis, Missouri

Carcinoma of the Cervix Uteri

The application of roentgen rays, radium, and other radioactive media in the treatment of carcinoma of the cervix will be described, and a clinical evaluation of the procedures will be made on the basis of: (1) the simplicity of the technique; (2) the flexibility of the method; (3) the reliability of the procedure; (4) the uniformity of the radiation intensity; (5) the control of the sources of radium; (6) the correction of uterine displacements and vaginal deformities; (7) the protection of the bladder and rectum; (8) the protection of operators and assistants; (9) the measurements of predetermined depth dose A/B coefficient in gamma roentgens; (10) the consistent duplication of the dose of radium in the same patient and in other patients by different therapists.

Other topics: (1) planning the clinical approach; (2) indications and contraindications; (3) selection of the method, type, form and timing of the radiation procedures; (4) external and transvaginal roentgen therapy; (5) discussion and demonstration of the more recent advances in technique based on measurements, with scintillation counters, of the distribution of the intensity of the radiation throughout the pelvis in gamma roentgens.

COURSE 502

Room B **Period: W**

CHARLES L. MARTIN, M.D.
Dallas, Texas

Treatment of Carcinoma of the Mouth and Secondary Metastatic Nodes with Irradiation

A large number of cases treated in a somewhat unorthodox manner will be discussed. All of the primary lesions and the metastatic nodes, when present, are implanted with low intensity radium needles, and roentgen therapy is used over the cervical areas when indicated. Carcinomas of the tongue, cheek, gums, floor of the mouth, palate, and accessible portions of the pharynx treated in this manner will be demonstrated with the aid of colored lantern slides. Surgical procedures are reserved for complications and radiation failures, and very radical procedures are seldom found necessary. A series of 94 consecutive cases of cancer of the tongue with an absolute five year cure rate of 32 per cent will be presented. No block dissections were carried out in this group of patients.

COURSE 503

Room C **Period: T**

J. A. del REGATO, M.D.
Colorado Springs, Colorado

Radiotherapy of Carcinoma of the Skin

Both surgery and radiotherapy may be effective in the treatment of carcinoma of the skin but radiotherapy has qualities, beyond the mere curability, which are not possessed by surgery.

Dependent on the situation and the extent of the lesion, the factors of kilovoltage, filtration and protraction of the treatment may be varied to advantage in order to best serve the purposes of control with the minimum of dysfunction or impairment.

When radiotherapy is properly administered, even very advanced cases of carcinoma of the skin can be locally controlled.

COURSE 504

Room C **Period: Th**

J. A. del REGATO, M.D.
Colorado Springs, Colorado

The Technique of Roentgen Therapy of Cancer of the Cervix

There is much to be gained by exhausting the pos-

sibilities of external roentgen therapy in the treatment of cancer of the cervix in all stages of development. A plan for this pelvic irradiation will be outlined.

The advantages and the rationale of the association of external roentgen therapy with transvaginal roentgen therapy will be presented.

COURSE 505

Room E **Period: Th**

ISADORE MESCHAN, M.D.
Little Rock, Arkansas

Dosimetry Problems in the Treatment of Cancer of the Cervix, with Special Reference to Radium and Cobalt 60

1. Emphasis upon the importance of conversion of all dosage data to gamma roentgens (estimated) as delivered to certain significant points in the pelvis.
2. The anatomic designation of such areas and points.
3. The description of a method whereby ready calculation is possible from any fixed applicator in the pelvis.
4. A method of charting the data so that calculation becomes relatively simple.
5. Discussion of ideal distribution of radiation in the pelvis, and the limiting factors, both biologic and geometric.
6. Description of an applicator of original design for use particularly with cobalt 60, which embodies a modification of the Manchester techniques.
7. Description of correlative histologic and gross appearances of the tissues of the pelvis in 26 operated cases, following the delivery of specified doses in specified times.
8. A brief analysis of the over-all problem as we see it now from the standpoint of how and where improvement in results is most likely to be obtained.

COURSE 506

Room E **Period: F**

ISADORE MESCHAN, M.D.
Little Rock, Arkansas

The Utilization of Cobalt 60 for Interstitial, Intracavitary, and Plaque Methods of Radiation Therapy

1. Presentation of basic physical data regarding the production of cobalt 60 and its disintegration.
2. Absorption data.
3. Conversion to roentgen dosimetry.
4. Presentation of tables for conversion to gamma roentgens from millicurie hours.
5. Presentation of modified Paterson-Parker tables.
6. Demonstrations of the utilization of such tables.
7. Summary of the biological testing of these when applied to rabbits' skin.
8. Presentation of example cases and results.

COURSE 507

Room J **Period: Th**

H. B. ELKINS, M.D.
Iowa City, Iowa

The Clinical Use of Au^{198}

The experience accumulated in using Au^{198} in over 300 cases in the past thirty months will form the basis for this course. Particular emphasis will be placed on common mistakes we have made and the manner in which they were corrected. The dose, protection of patients and personnel, the method of injection, complications, and results will be discussed.

COURSE 508A

Room L **Period: T**

HUGH F. HARE, M.D.
Los Angeles, California
and
JOHN G. TRUMP, Sc.D.
Cambridge, Massachusetts

Rotational Therapy Using 2 Million Volt Roentgen Rays: Physical Aspects

The course is divided into two parts. Included in this part will be a presentation of (1) the techniques of rotation therapy, (2) the distribution of dose in the irradiation of sites of typical tumors, (3) the physical and biological properties of roentgen rays over a wide range of energy with emphasis on 2 million volt rays, (4) consideration of the total body dose and fractionation techniques.

COURSE 508B

Room L **Period: Th**

HUGH F. HARE, M.D.
Los Angeles, California
and
JOHN G. TRUMP, Sc.D.
Cambridge, Massachusetts

Rotational Therapy Using 2 Million Volt Roentgen Rays: Clinical Aspects

The clinical application of the principles developed in Course 508A will be presented in this session. The use and results of 2 million volt therapy with rotation on tumors of the pituitary gland, nasopharynx, larynx, lymph nodes of the neck, esophagus, breast, lung, and pelvic cavity will be presented and discussed in as much detail as the time permits.

COURSE 601

Room C **Period: W**

JOHN F. HOLT, M.D.
Ann Arbor, Michigan

Roentgenologic Characteristics of Bone Tumors

This course consists of a review of certain basic problems in the roentgenologic diagnosis of bone tumors and related lesions illustrated by current case material. Emphasis will be placed upon pitfalls in differential diagnosis, the confusion of nomenclature as it now exists, and an appraisal of more recently described entities within this field.

COURSE 602

Room C **Period: F**

JACK FRIEDMAN, M.D.
Minneapolis, Minnesota

The Roentgenologic Approach to the Study of Emotional Disturbances in the Alimentary Tract

The functional effects of emotional distress observed in patients during roentgen examination is generally known to radiologists. The variable functional effects complicating roentgen examinations, primarily intended to seek out organic lesions of the gastrointestinal tract, will be discussed and evaluated. Review of the work of investigators in this area other than radiology will be correlated with the roentgen problems.

More intensive attention will be given to some recent roentgen studies attempting to demonstrate the effects of emotional distress on the small intestinal mucosal pattern. Case studies presented will demonstrate the cause and effect relationship of emotional distress to disordered motor function of the small intestine.

COURSE 603

Room D **Period: T**

C. ALLEN GOOD, M.D.
Rochester, Minnesota

Fungus Diseases of the Lungs

The roentgenologic manifestations of the various fungus diseases which affect the lungs are seldom sufficiently characteristic to justify a specific diagnosis. They may resemble those produced by tumors, by tuberculosis, or by many other inflammatory processes.

The variety of roentgenologic patterns produced by coccidioidomycosis, histoplasmosis, actinomycosis, nocardiosis, and blastomycosis will be discussed and examples shown. Other mycoses will be mentioned briefly.

COURSE 604

Room D **Period: W**

SVEN ROLAND KJELLBERG, M.D.
Stockholm, Sweden

Urological Problems in Pediatrics

About 400 urographic examinations and 600 urethrocystographic examinations constitute the experience upon which this discussion was founded. The urographic medium was a substance of the same chemical nature as diodrast in 35 per cent to 50 per cent concentration, or urokon in 30 per cent concentration. In practically all instances the urographic medium was injected intravenously. A special type of tilting table was used. There were no adverse reactions.

A special wing was constructed for use in urethrocystography. It was constructed in such a manner that the patient sat before a roll-film camera while voiding urine.

Roentgenograms were taken in frontal and lateral projections simultaneously, one to three seconds apart, until micturition had taken place. This method was used in children more than three years old; in younger children the roentgenograms are made in the prone position, the roentgen-ray beam in vertical direction. The important anomalies and pathological changes will be shown.

COURSE 605

Room D **Period: Th**

EDWARD B. D. NEUHAUSER, M.D.
Boston, Massachusetts

The Roentgen Diagnosis of Congenital Cardiovascular Abnormalities Amenable to Surgery

Many forms of congenital malformation of the heart and great vessels, long considered of academic interest only, are now surgically correctible. Recent advances in roentgen diagnosis and in surgical techniques are leading to the recognition of abnormalities which have been overlooked in the past, and have led to the more accurate diagnosis of many malformations whose major features have long been known.

The roentgen features of patent ductus arteriosus, coarctation of the aorta, stenosis of the pulmonary artery, tetralogy of Fallot, Corvisart's complex, double aortic arch, vascular rings constricting the trachea and esophagus, and anomalies of the right subclavian artery and of the innominate vein should be familiar to all roentgenologists. The diagnostic criteria of these malformations will be discussed.

COURSE 606

Room D **Period: F**

EDWARD B. D. NEUHAUSER, M.D.
Boston, Massachusetts

Certain Aspects of the Gastrointestinal Tract in Infants and Young Children

In this course, no effort will be made to cover all the possible abnormalities and diseases that are seen or which reflect themselves in abnormalities of the alimentary tract.

Particular attention will be given to the technique of examination of the esophagus and gastrointestinal tract in infants and in young children and also to the technique of examination of the colon in infants.

Particular attention will be devoted to congenital anomalies and resulting diseases of the esophagus, certain unusual forms of herniation of the bowel into the thorax, and considerable attention will be given to the problems of duplication of the alimentary tract.

Malrotation of the bowel, congenital stenosis and atresia and study of the patient with imperforate anus will be discussed.

Several other anomalies and acquired lesions will be discussed briefly.

COURSE 607

Room F **Period: W**

FRED O. COE, B.S., M.D.
Washington, D.C.

Cephalopelvimetry

A method of cephalopelvimetry is presented which possesses certain advantages not found in other methods.

The method is exact for pelvimetry.

The size and position of the skull can be determined in both cephalic and breech presentations.

The method is simple and easy for the technician.

Illustrative cases will be shown with a summary of the follow-up on outcome of delivery and measurements of infant skulls.

COURSE 608

Room F **Period: Th**

RICHARD H. MARSHAK
New York, New York

Roentgenologic Diagnosis of Diffuse Lesions of the Small Intestine

1. Localized and diffuse inflammatory lesions of the small intestine.
2. Diffuse non-inflammatory disorders of the small intestine including sprue, allergy, Whipple's disease, pellagra, Hodgkin's disease, hypoproteinemia, lymphosarcoma, etc.

The roentgen changes frequently encountered in the above conditions are increased secretions, dilatation, thickened folds, and segmentation. Numerous nonspecific terms are used to designate these changes. Study of a large number of patients with sprue indicates that these changes are best illustrated in this condition. The findings in sprue will be presented in detail together with an analysis of the above roentgen findings. Differential diagnosis from the numerous lesions mentioned above will be discussed.

3. Technique: Various preparations of barium sulfate; the effect of drugs.

COURSE 609

Room F **Period: F**

MAURICE D. SACHS, M.D.
Cleveland, Ohio

Cholangiography

Cholangiography serves a variety of useful purposes: (a) the detection of calculous and non-calculous obstructions or potential obstructions of the common duct at a risk appreciably lower than the standard surgical exploration; (b) the diagnosis of neoplastic and non-neoplastic disease of the pancreas; (c) anatomical orientation, especially in secondary operations on the biliary tract.

The conduct and technique of operative and postoperative cholangiography will be described in detail. This will include a discussion of various cholangiographic media, and of the surgical and roentgenologic equipment needed to perform the examination in a satisfactory way.

The normal anatomy of the biliary tract will be described with some comments on some of the accepted variations from the normal.

Among the pathological states considered will be: biliary calculi, spasm and inflammation of the sphincter of Oddi, acute and chronic pancreatitis, neoplastic involvement, postoperative structures of the common duct, choledochitis, and polypoid lesions.

COURSE 610

Room G **Period: T**

HAROLD O. PETERSON, M.D.
St. Paul, Minnesota

The Normal Skull

This will be a detailed review of the pertinent and practical aspects of the roentgenologic appearance of the normal skull. Interesting anatomic features, including normal variations and artefacts will be discussed in detail. At all times emphasis will be placed on a practical approach to the interpretation of standard roentgenographic projections of the skull. A few ideas on the roentgenographic technique will also be included.

COURSE 611

Room G **Period: W**

HAROLD O. PETERSON, M.D.
St. Paul, Minnesota

The Abnormal Skull

In this session the abnormalities of the skull which can be recognized on plain films will be discussed and illustrated. Again a practical everyday approach will be stressed.

COURSE 612

Room G **Period: Th**

HAROLD O. PETERSON, M.D.
St. Paul, Minnesota

Normal and Abnormal Cerebral Pneumography

Encephalography and ventriculography will be considered together. There will be no discussion on technique other than to illustrate the positions used in the routine examination. Minimum essentials of neuroanatomy will be stressed. Confusing patterns which occur frequently and are actually normal will be illustrated and explained. There will be a few general remarks on atrophic lesions. The fundamental deformities and displacements caused by masses will be shown and discussed.

COURSE 613

Room G **Period: F**

HAROLD O. PETERSON, M.D.
St. Paul, Minnestoa

Spinal Myelography

This course will be divided into two parts. The first part will consist of a detailed description of the technique of pantopaque or lipiodol myelography primarily from the standpoint of doing the spinal puncture with the aid of fluoroscopic control so that the contrast medium can be properly injected and readily and completely aspirated. Minute details and tricks learned over a period of years will be discussed.

The second part of the course will deal with interpretation and differential diagnosis. Particular emphasis will be placed on the value of myelography in the diagnosis of herniated intervertebral discs. Deformities which are pathognomonic will be illustrated and contrasted with those which are not. The localization of spinal cord tumors will be covered as completely as possible. There will also be a demonstration on the recognition of subdural extra-arachnoid injections of contrast media as compared with subarachnoid and extradural injections.

COURSE 614

Room H **Period: T**

A. S. MACMILLAN, M.D.
Boston, Massachusetts

Roentgen Examination of the Sinuses

1. Roentgen examination of the sinuses is a very important part of the diagnosis of pathology in these locations. The rhinologist is dependent upon a correct interpretation of the sinus roentgenograms. The findings should be reported in terms of pathology, and not in the indefinite roentgenological terms so often used. In this presentation, the technique of examination and interpretation will be considered as fully as time will allow.

COURSE 615

Room H **Period: W**

A. S. MacMILLAN, M.D.
Boston, Massachusetts

Roentgen Examination of the Mastoids

2. The use of chemotherapy and antibiotics has greatly changed the course of infection in the mastoid. Pediatricians and general medical practitioners sometimes feel competent to treat this condition, but too often the treatment is inadequate. The roentgenologic examination of the mastoid is the only dependable source of information. This should be recognized because mastoiditis is still prevalent. Acute and chronic infections of the mastoid and the complications will be discussed.

COURSE 616

Room H **Period: Th**

A. S. MacMILLAN, M.D.
Boston, Massachusetts

Film Reading Session

3. *The film reading session* will consider conditions met in a hospital devoted to treatment of the sinuses, mastoids and abnormalities of the esophagus. Rare and unusual conditions are often emphasized in a presentation such as this, with failure to give adequate consideration to the more common types of pathology met with every day. This discussion will deal with the more commonly encountered abnormalities in which roentgenologic assistance is sought.

COURSE 617

Room H **Period: F**

ARTHUR T. EVANS, M.D.
Cincinnati, Ohio

Translumbar Arteriography

Translumbar arteriography has a valuable contribution to make to urologic diagnosis. In this course the technique, the indications and contraindications, the dangers, the timing, and the specific advantages of the procedure will be discussed in considerable detail. A moving picture, supplemented with lantern slides, will be used to illustrate the points that will be made.

COURSE 618

Room I **Period: Th**

DAVID G. PUGH, M.D.
Rochester, Minnesota

The Roentgenologic Diagnosis of Arthritis

There are two basic problems in the diagnosis of arthritis. The first is to determine if arthritis is present. The second is to distinguish the various types of arthritis. In this discussion on the roentgenologic diagnosis of arthritis we will first investigate the structures in the joint that are affected and note the ways in which alteration in these structures manifest themselves roentgenologically. We will then consider the manner in which various types of arthritis affect joints. This has to do especially with the number and location of the joints affected, and the extent and rapidity of the destructive process. With these factors in mind it is possible to recognize arthritis and identify the type with considerable accuracy.

COURSE 619

Room J **Period: T**

ROBERT N. COOLEY, M.D.
Baltimore, Maryland

The Use of Angiocardiography and Thoracic Aortography in the Diagnosis of Congenital Cardiovascular Malformations

The first portion of the period will be spent in describing the technique of angiocardiography and aortography. Such aspects as preliminary medication, anesthesia, contrast substances, automatic injectors, film changing devices and the hazards of the procedures will be discussed in considerable detail. Particular emphasis will be placed on those modifications which are useful in children and infants.

The second portion of the period will be given to a description of the angiocardiographic and aortographic appearance of the more common congenital anomalies. A series of proven cases will be shown in order to evaluate the reliability and accuracy and the shortcomings of the procedure.

COURSE 620

Room J **Period: W**

ROBERT L. SCANLAN, M.D.
Philadelphia, Pennsylvania

The Roentgen Diagnosis of Gallbladder and Biliary Tract Disease without Cholecystography

A great deal of information concerning abnormalities of the gallbladder and biliary tract is available on conventional roentgenograms, without the advantage of cholecystography. Films of the abdomen frequently reveal biliary tract disease, often unsuspected and significant. Stones in the gallbladder and biliary ducts, distended gallbladder, and air in the biliary tract are not uncommonly demonstrated, particularly in acute abdominal disease, and when the gallbladder is not visualized on the cholecystogram.

In this course, common and unusual findings will be illustrated and the technique of abdominal roentgenography will be emphasized.

COURSE 621

Room J **Period: F**

H. F. PLAUT, M.D.
Dayton, Ohio

Vertebral Arteriography

1. The normal anatomy of the vertebral arteries and their branches is described. Examples of anomalies and variations are explained as remnants of embryonic stages (slides).
2. Indications for vertebral arteriography are mentioned in suspected vascular lesions and neoplasms of the regions supplied by the system of the vertebral arteries.
3. After a short discussion on the different techniques the normal vertebral arteriogram is shown as performed on the living and the cadaver.
4. The roentgenographic signs in pathologic conditions are illustrated.

COURSE 622

Room L **Period: W**

W. E. BAENSCH, M.D.
Washington, D.C.

Cinematography of the Angiocardiogram of the Normal Heart and Congenital Diseases of the Heart

The cinematography was done by Dr. Janker.

Schematic drawings are shown in all cases before demonstration in the pictures. The presentation starts with a moving picture of the normal heart. At first, the dextrogram is demonstrated with filling through the superior vena cava. This is followed by demonstration of the levogram. Then, angiocardiograms of three normal patients with normal pulsation are shown, repeated in slow motion so that all details can be observed.

In the second part, moving pictures of angiocardiograms of the most common congenital diseases of the heart are demonstrated. The angiocardiogram seen in tetralogy of Fallot, characterized by pulmonary stenosis, ventricular septal defect, overriding of the aorta, and hypertrophy of the right ventricle is shown. Schematic drawings show the characteristics of this disease. The moving picture of the angiocardiogram of a patient with typical Fallot syndromes is demonstrated. The motion on this film is five times slower than normal.

The second moving picture shows the Eisenmenger complex. It is followed by angiocardiograms in cases of truncus arteriosus communis, tricuspid stenosis, pulmonary stenosis, coarctation of the aorta, and patent ductus.

The diagnosis of all cases presented was proved by operation. The typical changes in the dextrogram and levogram in these most common congenital diseases of the heart will be demonstrated.

CONDENSED SCHEDULE OF COURSES ON TUESDAY, SEPT. 29, 1953

Code: T 3:00–4:30 P.M. **Code: T**

Therapeutic Radiology, Radiation Physics, Miscellaneous Subjects		Diagnostic Roentgenology	
501—Ernst Room B	Carcinoma of the Cervix Uteri	**401A—MacCarty and McLaughlin Room E**	Excretory Urography Part I
503—del Regato Room C	Radiotherapy of Carcinoma of the Skin	**603—Good Room D**	Fungus Diseases of the Lungs
508A—Hare and Trump Room L	Rotational Therapy using 2 Million Volt Roentgen Rays: Physical Aspects	**610—Peterson Room G**	The Normal Skull
202—Hodges Room F	The Library of Teaching Films	**614—Macmillan Room H**	Roentgen Examination of the Sinuses
101—Corrigan and Panel Room I	Use of Radioisotopes in Diagnosis	**619—Cooley Room J**	Angiocardiography and Thoracic Aortography in Diagnosis of Congenital Cardiovascular Malformations

CONDENSED SCHEDULE OF COURSES ON WEDNESDAY, SEPT. 30, 1953

Code: W — **3:00–4:30 P.M.** — **Code: W**

Therapeutic Radiology, Radiation Physics Miscellaneous Subjects		Diagnostic Roentgenology	
303—Morgan Room A	The Place of Electronics in Roentgenology	**601—Holt Room C**	Roentgenologic Characteristics of Bone Tumors
502—Martin Room B	Treatment of Carcinoma of the Mouth and Metastatic Nodes with Irradiation	**604—Kjellberg Room D**	Urological Problems in Pediatrics
102—Corrigan and Panel Room I	Use of Radioisotopes in Therapy	**401B—MacCarty and McLaughlin Room E**	Excretory Urography Part II
		607—Coe Room F	Cephalopelvimetry
		611—Peterson Room G	The Abnormal Skull
		615—Macmillan Room H	Roentgen Examination of the Mastoids
		620—Scanlan Room J	Roentgen Diagnosis of Gallbladder and Biliary Tract Disease without Cholecystography
		622—Baensch Room L	Cinematography of the Angiocardiogram of the Normal Heart and Congenital Diseases of the Heart

CONDENSED SCHEDULE OF COURSES ON THURSDAY, OCT. 1, 1953

Code: Th	3:00–4:30 P.M.	Code: Th

Therapeutic Radiology, Radiation Physics, Miscellaneous Subjects		Diagnostic Roentgenology	
302—Parker Room A	Radiation Dosimetry	**402A—Dotter Room B**	Angiocardiography Part I
504—del Regato Room C	Technique of Roentgen Therapy of Cancer of the Cervix	**605—Neuhauser Room D**	Roentgen Diagnosis of Congenital Cardiovascular Abnormalities Amenable to Surgery
505—Meschan Room E	Dosimetry Problems in the Treatment of Cancer of the Cervix, with special reference to Radium and Cobalt 60	**608—Marshak Room F**	Roentgenologic Diagnosis of Diffuse Lesions of the Small Intestine
507—Elkins Room J	The Clinical Use of Au^{198}	**612—Peterson Room G**	Normal and Abnormal Cerebral Pneumography
508B—Hare and Trump Room L	Rotational Therapy using 2 Million Volt Roentgen Rays: Clinical Aspects	**616—Macmillan Room H**	Film Reading Session
		618—Pugh Room I	Roentgenologic Diagnosis of Arthritis

CONDENSED SCHEDULE OF COURSES ON FRIDAY, OCT. 2, 1953

Code: F 1:30–3:00 P.M. **Code: F**

Therapeutic Radiology, Radiation Physics, Miscellaneous Subjects		Diagnostic Roentgenology	
301—T. C. Evans Room A	Fundamental Aspects of the Effects of Ionizing Radiation	**402B—Dotter Room B**	Angiocardiography Part II
506—Meschan Room E	Utilization of Cobalt 60 for Interstitial, Intracavitary, and Plaque Methods of Radiation Therapy	**602—Friedman Room C**	Roentgenologic Approach to the Study of Emotional Disturbances in the Alimentary Tract
201—W. E. Chamberlain Room I	Radiologic Ethics, Administration and Economics	**606—Neuhauser Room D**	Certain Aspects of the Gastrointestinal Tract in Infants and Young Children
		609—Sachs Room F	Cholangiography
		613—Peterson Room G	Spinal Myelography
		617—A. T. Evans Room H	Translumbar Arteriography
		621—Plaut Room J	Vertebral Arteriography

FIFTH ANNUAL CANCER SEMINAR

The Fifth Annual Cancer Seminar will be held at the Broadmoor Hotel, Colorado Springs, Colorado, September 5, 1953. The subject of this year's Cancer Seminar will be "Lesions of the Small Bowel." The guest radiologist will be Dr. Paul C. Swenson, Professor of Radiology at Jefferson Medical College, Philadelphia, and the guest pathologist will be Dr. William A. Meissner of the Deaconess Hospital of Boston. Dr. Harry M. Weber of the Mayo Foundation, Rochester, Minnesota, will be the guest of honor.

The Cancer Seminar is sponsored by the Penrose Cancer Hospital and by the American College of Pathologists.

RADIOLOGICAL HEALTH TRAINING PROGRAM

A series of short courses in radiological health are again being presented by the Public Health Service at the Environmental Health Center in Cincinnati, Ohio. These courses, tuition free, are designed primarily to provide professional personnel working in health departments and other organizations with a working knowledge of the health hazards associated with radiation. Candidates should have a degree in medicine, engineering or science (physical or biological).

Instruction is given in radiation physics sufficient for an understanding of the problems arising from proper and improper use of ionizing radiation. Laboratory and lecture periods are presented covering the hazards, and methods of alleviating these hazards, as encountered in employment of roentgen rays, radioisotopes, neutron fluxes and particle accelerators.

Courses are each two weeks in length. Basic Courses, which stress basic radiation physics and survey techniques, will be presented: October 5–16, 1953; January 11–22, 1954; May 3–14, 1954.

The Basic Course or its equivalent is a prerequisite for the Intermediate Course. Laboratory assay of radioelements and roentgen-ray survey techniques are emphasized in the Intermediate Course which will be presented: October 19–30, 1953; January 25–February 5, 1954; May 17–28, 1954.

Completion of the Intermediate Course or its equivalent is a prerequisite for admission to the Advanced Course. This course, for professional personnel concerned in particular with occupational health problems, will be presented on February 8–19, 1954.

Further details and information may be obtained by writing: Chief, Radiological Health Training Section, Public Health Service, Environmental Health Center, Cincinnati, Ohio.

TELETHERAPY AND RADIOGRAPHIC PROBLEMS WITH ISOTOPES

There will be a joint meeting of the Oak Ridge Institute of Nuclear Studies and the Isotopes Division with all interested members of the X-ray Industry on teletherapy and radiographic problems with isotopes. The purpose of the meeting, which is to be held on October 4 and 5, 1953, is to informally review the current thinking on teletherapy design problems, to review the protection requirements for large sources, to discuss the possibilities of the standardization of large source encapsulation, and to review the present status of source manufacture and procurement. All members of the X-ray Industry are invited to participate. More detailed information can be obtained fron the Medical Division, Oak Ridge Institute of Nuclear Studies, Oak Ridge, Tennessee.

SYMPOSIUM ON FUNDAMENTAL CANCER RESEARCH

Charles B. Huggins, M.D., received this year's Bertner Foundation Award at the Seventh Annual Symposium on Fundamental Cancer Research which was held May 15 and 16, 1953, at the Shamrock Hotel, Houston, Texas. Conferred annually on a physician or scientist for having made an outstanding contribution to some field of cancer research, the award was established in 1950 in honor of the late Dr. E. W. Bertner, first acting director of The University of Texas M. D. Anderson Hospital for Cancer Research, and the first

president of the Texas Medical Center in Houston. The 1953 award was presented to Dr. Huggins, who is professor of surgery at the University of Chicago School of Medicine, for his classic studies in the field of controlling human cancer by physiologic methods.

Dr. Huggins also delivered the annual Bertner Lecture, in which he outlined the history of his work, and revealed that 20 per cent of his original series of patients—males with prostatic cancer—were in good health after having been castrated thirteen years ago.

Dr. Huggins further outlined his work in a paper, "The Steroid-Dependent Mammary Cancer," which was one of the twenty papers delivered during the two-day session attended this year by approximately 250 physicians, scientists and laymen from sixteen states and Mexico.

Conducted by the Anderson Hospital, the annual Symposium is held jointly with a Cancer Pathology and Radiology Conference in which the South Central Region College of Pathologists, Texas Society of Pathologists and Houston Society of Pathologists are co-participants.

The chief topic of this year's Symposium was "Newer Methods of Microscopy and Cell Research," with a program of five speakers arranged by Robert C. Mellors M.D., Ph.D., Division of Experimental Pathology and Pathology Laboratories, Memorial Center, New York.

Edgar Altenburg, Ph.D., associate professor of Biology, Rice Institute, Houston, was chairmen of a general session in which six more papers were presented by cancer researchers, and May Owen, M.D., Fort Worth, Texas, was chairman of a joint session in which the pathologists took part on the closing day.

Guest conductors of the Pathology and Radiology Conference were David A. Wood, M.D., professor of oncology and director of the Cancer Research Institute at the University of California School of Medicine, and W. Edward Chamberlain, M.D., professor of radiology at Temple University Hospital, Philadelphia.

The conference was devoted to "Tumors of the Lung and Pleura," with William O. Russell, M.D., pathologist in chief at Anderson Hospital as the moderator. Attendance included 64 pathologists and 24 radiologists.

The program was co-ordinated with the Anderson Hospital's monthly Educational Slide Set Service, to which nearly 150 pathologists in the eight-state territory of the South Central Region regularly subscribe.

For the joint meeting, approximately 225 sets of slides, and more than 800 syllabi of 12 case histories, clinical findings, etc., were sent out.

Concluding the two-day session, John R. McDonald, M.D., professor of pathology at the Mayo Foundation Graduate School was chairman of a critique of cytological programs in which two pathologists, two surgeons, a radiologist and a gynecologist participated.

CHICAGO ROENTGEN SOCIETY

The following are the newly elected officers of the Chicago Roentgen Society to serve from April, 1953, to April, 1954: *President*, Dr. Roger A. Harvey; *Vice-President*, Dr. Erich Uhlmann; *Secretary-Treasurer*, Dr. Elbert K. Lewis, all of Chicago, Illinois.

LOS ANGELES RADIOLOGICAL SOCIETY

At the annual election of the Los Angeles Radiological Society held in May, 1953, the following officers of the Society were elected to take office in September, 1953 for the year 1953–1954: *President*, Dr. Joseph F. Linsman; *Vice-President*, Dr. M. M. Haskell; *Secretary*, Dr. George Jacobson; *Treasurer*, Dr. Hubert J. Prichard; *Executive Committee*, Dr. Harold P. Tompkins, Dr. John B. Hamilton.

NEW ENGLAND ROENTGEN RAY SOCIETY

At the annual meeting of the New England Roentgen Ray Society held at the Hotel Somerset on Friday, May 15, 1953,

the following officers were elected to serve for 1953–1954: *President*, Dr. Laurence L. Robbins; *Vice-President*, Dr. Alice Ettinger; *Secretary*, Dr. Stanley M. Wyman; *Treasurer*, Dr. Magnus I. Smedal, all of Boston, Massachusetts. The Holmes Annual Lecture was presented by Dr. Edith H. Quimby of New York and the subject was "Diagnostic Uses of Radioactive Isotopes."

PHILADELPHIA ROENTGEN RAY SOCIETY

At the May meeting of the Philadelphia Roentgen Ray Society the following were elected to office for the year 1953–1954: *President*, Dr. George P. Keefer; *Vice-President*, Dr. Rolfe Harvey; *Secretary*, Dr. Herbert M. Stauffer; *Treasurer*, Dr. D. Alan Sampson.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the May meeting of the Radiological Society of New Jersey, the following officers were elected for the year 1953–1954: *President*, Dr. Vincent Whelan; *Vice-President*, Dr. Nicholas G. Demy; *Secretary*, Dr. Salmon Silvera; *Treasurer*, Dr. C. B. Henle.

Vol. 70, No. 2

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Vogel, F. Stephen, and McClenahan, John L. Anomalies of major cerebral arteries associated with congenital malformations of the brain; with special reference to the pathogenesis of anencephaly. *Am. J. Path.*, July-Aug., 1952, *28*, 701–723.

It has been known that congenital anomalies of the central nervous system, including anencephaly, can be induced by means of anoxia, irradiation by roentgen rays, and by alterations in the thermal, osmotic, or ionic environment of the developing embryo. The nature of the malformation seems to depend more upon the stage of embryonic development at the time of the experiment than it does upon the means by which the injury is induced. To learn about pathogenesis of congenital malformation in the brains of human beings, detailed postmortem examinations were performed on 14 anencephalic monsters. Particular attention was paid to the cerebral arterial system and an original injection technique was devised for a more accurate study of the cerebral blood vessels. In addition, alterations were induced in the brains of developing chick embryos by occluding one or another of the major cerebral arteries, and these were studied in relation to the naturally occurring congenital anomalies of the brain.

To study the blood supply of anomalous brains, the vessels were injected with a fusible metal which hardens upon cooling and which can be studied in situ by means of roentgenography or by examination of the casting which remains after digestion of the tissues with alkali. This technique was used in a study of 5 cases of anencephaly, in each of which some anomaly of the major cerebral arteries was demonstrated. This raised the question whether a specific intrinsic structural alteration or morphological lesion might be present within the walls of the deformed cerebral arteries. Histopathological studies made thus far have failed, however, to disclose the nature of the vascular anomalies.

In the study of six day old chick embryos one or another of the cerebral arteries was occluded by electrocauterization. This procedure was regularly followed by a retardation of cerebral development and by alterations in the differentiation of the neuronal elements. A possibility must therefore be considered that vascular anomalies of the major cerebral arteries may be a factor in the pathogenesis of anencephaly in human beings.—*Paul R. Dirkse, M.D.*

Uhlmann, W. Über eine neue Kombinationsvariante von Dysostosis cranio-facialis mit Akrocephalosyndaktylie. (A new variant of craniofacial dysostosis with acrocephalosyndactyly.) *Fortschr. a. d. Geb. d. Röentgenstrahlen ver m. Röntgenpraxis*, Aug., 1952, *77*, 213–219.

A case of Crouzon's craniofacial dysostosis, showing the typical brachycephalic turricephaly, with marked prominence of the bregma, exophthalmos with divergent squint and partial blindness, antimongoloid obliquity of the palpebral fissures, hypoplasia of the maxillas associated with a short upper lip and a highly arched palate, and prognathism of the mandible, was found to have synostosis of the distal row of carpal bones bilaterally, bilateral fusion of the calcaneus and cuboid, and partial fusion of the navicular and first cuneiform bones. This occurred despite the absence of any actual syndactyly such as that described by Apert in similar cases. Fusion of the lower three cervical vertebrae was also associated. Skull roentgenograms showed closure of all the sutures of the vault, prominent convolutional markings, a widened and deepened sella turcica, shallow orbits, basilar impression, and a prominent intermaxillary suture, ascribed to hypoplasia of the premaxillary bones. The patient was partially deaf as well as partially blind, of limited intelligence, with a cheerful child-like disposition, and suffered from headaches that might be attributed to episodes of increased intracranial pressure, but had no convulsive episodes.

Bonnevie, seeking a unitary origin for the various defects, from observations on mouse embryos, states that in an early stage of development there is normally a hypersecretion of cerebrospinal fluid, which leaks from the physiological opening in the 4th ventricle, and spreads in the subcutaneous tissues to the sites of least resistance, the nose, orbits, and the anlage of the hands and feet, forming actual vesicles. Normally these disappear, but if hypersecretion continues, they persist longer, and lead to the anomalies described.—*John J. B. Light, M.D.*

Ituarte, R. Bueno. Un caso de enfermedad de Pick. (A case of Pick's disease.) *Bol. Inst. Pat. Méd.*, Sept., 1952, *7*, 165–167.

In 1892, Pick described a disease characterized by a circumscribed atrophy of the cerebral cortex, particularly of the frontal or temporal lobe, and more rarely of the parietal lobe. The disease begins in the fifth or sixth decade of life, or even earlier, and in a few years marked alteration of personality leads to dementia and death. Today it is considered as a disease of premature senile changes, at times hereditary, of the cerebral parts cited above.

A sixty-four year old male presented himself with onset of vague cephalic and nervous symptoms of five years' duration. Personality alteration had occurred. Pneumoencephalography revealed dilated ventricles with great accumulation of air in the subarachnoid spaces, particularly in the frontal region. Later, loss of inhibitions and deterioration of

judgment progressed.

It is difficult, at times, to differentiate Pick's disease from another presenile dementia, the disease of Alzheimer. In the latter, the psychic deficit is more global, in spite of which affective faculty is well conserved.—*Charles M. Nice, M.D.*

Cant, W. H. P., and Astley, R. Lipoma of the corpus callosum. *Arch. Dis. Childhood*, Oct., 1952, *27*, 478–479.

The authors report a case of lipoma of the corpus callosum in a five year old male that was associated with a congenital lipoma of the scalp. Nothing unusual was found on physical examination but there was a history of a generalized convulsion.

The following roentgenological features are characteristic: (1) the curved plaques of calcification, symmetrically arranged on each side of the midline in the region of the corpus callosum; (2) the translucency of fat (although this may be obscured by calcification in the capsule); (3) the displacement of the lateral ventricles on each side of the midline mass demarcated by the calcification.

The anterior cerebral arteries are often incorporated in a lipoma of the corpus callosum. This anatomical relationship has led to permanent postoperative disorders of cerebral function or death in the 4 cases recorded where surgery has been attempted.

The lesion is a congenital malformation, with replacement of the corpus callosum by fatty tissue, rather than a true tumor. It presents no clear-cut clinical picture but the pathognomonic roentgen appearance may make diagnosis possible during life. This is important because surgical removal appears to be contraindicated.—*James F. Martin, M.D.*

Trufant, S. A., and Seaman, William B. Unilateral calcification of the basal ganglia. *Radiology*, Oct., 1952, *59*, 521–524.

A case of unilateral calcification of the basal ganglia of the brain, demonstrated roentgenologically, and associated with contralateral manifestations of extrapyramidal disease is described. Two excellent roentgenographic reproductions demonstrate the typical fan-shaped vertical striations in the region of the basal ganglia.

Typical contralateral neuromuscular symptoms which had progressed over the past fifteen years are described.

There is a short general discussion of intracranial calcification and a review of the literature regarding calcification of the basal ganglia.—*Everett E. Seedorf, M.D.*

Heppner, F. Air embolism eight hours after ventriculography; a case report. *Acta radiol.*, Oct., 1952, *38*, 294–298.

Eight hours after a ventriculography carried out in the typical parieto-occipital manner the patient suddenly died. Death was caused by air embolism. The lobe-shaped laceration of the venous wall probably kept the vessel shut like a valve during the ventriculography. A sudden increase of the venous pressure, perhaps by coughing, opened this valve. Because of the patient's position with the upper part of the body raised, the venous pressure was negative and the air contained in the ventricle was aspirated into the vein. Autopsy excluded the other possibility, *i.e.*, that communication between the circulatory and the ventricular system by the pathologically vascularized tumor had occurred.

The author concludes that ventriculography is indicated only when there is complete blockage of cerebrospinal fluid and where no ventricular filling can be obtained by means of encephalography. By the employment of ascending encephalography cerebral herniation is prevented.

Whenever patients with increased intracranial pressure cannot be operated upon immediately after ventriculography, a recumbent position in bed is recommended. In such a case the increase of intracranial pressure must be regarded as the lesser evil and be treated by osmotherapy rather than by raising the upper part of the patient's body in bed. Only in this way can the decrease in the intravenous tension to below zero be avoided and a possible intravenous aspiration of ventricular air prevented.—*Mary Frances Vastine, M.D.*

Naulleau, J., Poirier, R., and Abelanet, R. Adamantinome des deux maxillaires supérieurs. (Adamantimona of both maxillary bones.) *Presse méd.*, Oct. 22, 1952, *60*, 1440–1442.

This is a case report of an adamantinoma arising simultaneously from both maxillary bones, first diagnosed in 1950 at which time the patient refused operation. Considerable increase in size occurred during the following eight months and finally a subtotal resection of both maxillary bones was done. A small recurrence was detected three months later and treated with roentgen therapy (8,000 r delivered through three portals). The patient was well six months after the conclusion of treatment and a prosthesis fitted to cover the large bone defect.

In the discussion of this case, terminology and histology are first dealt with. The rarity of metastasis is stressed (4.5 per cent according to Robinson). There seems to be a predisposition of the colored races according to Geschickter and Copeland and Schulenberg. The localization in the maxilla is rare; in a series of 379 cases it was found in 16 per cent, the mandible being the most common location. The simutaneous involvement of both maxillary bones is still more unusual.

Anesthesia and surgery are then discussed and finally roentgen therapy. Adamantinomas have usually been considered as rather radioresistant,

but Baclesse and Letoue have recently reported a series of 14 cases with one four year cure in 2 cases treated by roentgen therapy only.

The paper contains reproductions of one roentgenogram and photographs of six gross and microscopic specimens.—*E. G. Wissing, M.D.*

Sergent, P., Rougerie, J., Pertuiset, B., and Petit-Dutaillis, D. L'angiographie vertébrale percutanée cervicale antérieure d'après 130 cas; technique et bases de l'interprétation des clichés, (Angiography of the vertebral artery by percutaneous anterior cervical injection, 130 cases; technique and basis for interpretation.) *Presse méd.*, Oct. 22, 1952, *60*, 1415–1418.

A short historical review of the different techniques and their development is given, beginning with that of Egas Moniz in 1933, and the variations in the surgical approach are described leading up to the percutaneous injection technique of Lindgren (1947). The authors have used the latter method. This technique has brought the examination within the everyday range of the radiologist.

The artery is punctured from anteriorly within the vertebral canal where it is immobilized by the surrounding bone and where there is no danger of damaging other structures. The space between C5–C6 or C4–C5 is used in order to avoid irritation of the nerve roots of the phrenic nerve. Injection is rapid and no general anesthesia is necessary. Sedatives and an antispasmodic are given. However, general anesthesia is used in children.

Preliminary roentgenograms of the cervical spine are made to rule out bone or joint lesions. The technique of the angiography follows closely Lindgren's description and is discussed in considerable detail. The authors use 5–8 cc. of 45 per cent diiodone and make three exposures during six seconds (one every two seconds), in the lateral, submento-occipital (Hirtz position) and basal (cerebellar) view.

The second half of the article deals with the anatomy of the vertebral artery and with the interpretation of the roentgenograms. It is emphasized that the morphology is variable and that the right and left vertebral arteries do not always appear symmetrical. The anatomy is given in great detail. The lateral view is the most important, the submento-occipital the least. The venous phase is also considered and the roentgenographic appearance in the lateral view showing the venous return is given together with a diagram. In conclusion, the difficulties of the method are stressed.

The paper is a detailed study of the technique and normal anatomy of the vertebral artery and its branches. There are excellent reproductions of roentgenograms and diagrams. The authors announce a further article dealing with the diagnostic application of the method and with indications for its use in lesions above and below the tentorium.—*E. G. Wissing, M.D.*

Psenner, L. Beitrag zur Klinik und zur Röntgendiagnostik des Chordoms der Schädelbasis. (On the clinical course and roentgenological diagnosis of chordoma of the base of the skull.) *Fortschr. a. d. Geb. d. Röentgenstrahlen ver m. Röntgenpraxis*, Oct., 1952, *77*, 425–433.

The author presents 2 cases of chordoma of the skull, 1 of them verified by autopsy.

The clinical symptoms are paralysis of cranial nerves, absence of increased intracranial pressure and often signs of hypophyseal tumor such as bitemporal hemianopsia or monolateral or homonymous hemianopsia. Pressure on the hypothalamus causes obesity, low basal metabolism, rise in blood pressure and frigidity in women. With extension of the tumor posteriorly, symptoms of a lesion of the cerebellum, the medulla oblongata and in the late stages symptoms of increased intracranial pressure appear. Pain and rigidity of the neck are observed with involvement of the foramen occipitale magnum. Protrusion into the nasopharynx and involvement of the orbit with exophthalmos are rare.

The first case, a forty year old female, complained of paralysis of the eye muscles and decreased vision in the right eye of several months' duration. Roentgen examination revealed destruction of the right anterior clinoid process, asymmetrical excavation of the sella, osteoporosis of the dorsum sellae and calcifications in the suprasellar region. Carotid arteriography and ventriculography showed a space occupying lesion in the sellar region so that parasellar meningioma was the tentative diagnosis. On operation severe bleeding from the tumor was encountered and removal was found impracticable. The patient died on the following day with symptoms of increasing intracranial pressure. At autopsy a large vascular chordoma of the sella with calcifications was found.

The second case, a forty-eight year old female, with a complete paralysis of the right oculomotorius of two months' duration, was referred for roentgen examination. Roentgenograms revealed an ill-defined destruction of the clivus involving the dorsum sellae and the posterior portion of the floor of the sella. The neurosurgeon considered the case inoperable and roentgen therapy was administered. A total dose of 5,800 r was given through a right and left lateral field. After 2,000 r the oculomotorius paralysis began to diminish and at the end of the treatment there was almost complete return to normal function. The area of destruction diminished and the dorsum sellae recalcified. The author assumes that the radiosensitivity was due to sarcomatous degeneration of the chordoma. No follow-up period is mentioned.—*Karol E. Matzinger, M.D.*

Neck and Chest

Callahan, William P., Jr., Sutherland, John C., Fulton, John K., and Kline, John R. Acute diffuse interstitial fibrosis of the lungs. *A.M.A. Arch. Int. Med.*, Oct., 1952, *90*, 468–482.

The authors add 1 case to the literature, bringing the total reported to 16. The present case is similar to previous ones symptomatically showing cough, dyspnea and hemoptysis. Histopathologically, two new findings were encountered in the lungs: First, in the alveolar septa, an increase in connective tissue and young fibroblasts, proliferation of young capillaries and interstitial edema. These changes were also noted in the perivascular and peribronchial spaces. In the lower lobes peripherally the alveolar septa were broken and large emphysematous pockets were formed. Secondly, the alveolar spaces were filled with fibrinous material. There were no inflammatory cells in the septa or transudate.

Four roentgenograms of the chest are presented. These were made on hospital days one, four, seventeen and twenty-two (the last, five days before death). These show rapidly worsening changes ranging from heavy peribronchial thickening at first to extension and diffuse changes in density at last. On all films varying degrees of pneumonic consolidation are present. Poor aeration is a notable feature.

The authors feel that with the physical findings of cough, dyspnea, hemoptysis and cyanosis plus the rapidly changing roentgenographic appearance in the absence of signs of infection the picture is sufficiently clear to warrant a clinical diagnosis. The paper discusses pathogenesis mentioning chronic lymphedema or possibly a virus as a causative factor.—*P. B. Parsons, M.D.*

Boucher, H., Delmas, J., and Mieral, R. Un syndrome radio-clinique peu connu: l'atélectasie en bande de Fleischner. (A little known clinical-roentgenological syndrome: Fleischner's band-like atelectasis.) *Presse méd.*, Oct. 22, 1952, *60*, 1437–1440.

The authors state in the introduction to their paper that the band-like (or disc-shaped) atelectasis, although described in 1936 by Fleischner, is still almost unknown in France. They then discuss briefly Fleischner's original paper and some of the work done by others following the original publication, particularly in regard to the increasing number of etiological factors.

The cases are classified according to their origin, either as abdominal or thoracic. The cases with subdiaphragmatic origin are due to paralysis of the diaphragm or limitation of its motility. Four of such cases are described due to trauma, liver biopsy or infection. One case is attributed to acute poliomyelitis. The other group of cases are of purely thoracic origin. Several of them are attributed to acute infectious pneumonia, virus pneumonia, etc. The frequency of these findings is again stressed in the summary.

The paper contains nineteen illustrations, most of them reproductions of roentgenograms, including several laminagrams and a photograph of a pathological specimen.—*E. G. Wissing, M.D.*

DeNardi, Joseph, M., Van Ordstrand, H. S., and Curtis, George, H. Berylliosis; summary and survey of all clinical types in ten year period. *Cleveland Clin. Quart.*, Oct., 1952, *19*, 171–193.

Readers who wish to delve further into the subject of berylliosis will find in this article nineteen key bibliographic references concerning the toxicology, clinical and experimental pathology, and industrial hygiene of this disease, plus a brief direct discussion of the offending occupations and compounds, admissible exposure, etc. This is a concise summarizing report of the Cleveland authors' unique volume of experience with dermal and pulmonary berylliosis between 1940 and 1952. They have encountered 202 cases with skin or eye symptoms, and 229 with respiratory tract disease during the twelve year period. Urine and blood analyses for beryllium have been possible since 1948 in only a fraction of the total group since the mass of cases occurred during and after the war years among migrant workers who are now lost to follow-up, and prophylactic measures instituted in recent years have reduced sharply the incidence of the clinical syndromes.

With reference to the spectrographic and fluorometric studies done upon urine, blood, biopsy or necropsy tissues of pulmonary patients, the data indicate (tentatively) that even symptom-free employes with chronic exposure will show significant traces of beryllium in the urine; that excretion of Be in the urine diminishes roughly in direct proportion to the length of time since last exposure. It also appears that control individuals, who have been exposed without developing pulmonary symptoms at any time, have essentially the same magnitude of excretion values as those who have had either the acute or chronic forms of pulmonary berylliosis. But in fatal cases of the two disease groups (acute and chronic), the acute pneumonitis patients have larger quantities of beryllium in necropsy specimens.

Acute beryllium tracheobronchitis did not cause any deaths in 129 patients, but acute pneumonitis was fatal in 11 per cent of 93 cases. After recovery from the acute type of disease, only 1 substantiated case went on later to develop chronic pulmonary granulomatosis (there may also be 2 others in the chronic group with histories suggesting previous acute tracheobronchitis). The authors seem to imply

that the individual who has had an acute manifestation is thereby statistically protected from subsequently developing chronic pulmonary disease. However, while they have follow-up studies in 50 per cent of their recovered acute pneumonitis cases to buttress this point, almost 75 per cent of the tracheobronchitis cases have been lost to follow-up. Since the latent period may be several years before exposed individuals develop the chronic disease, we cannot yet surmise the eventual incidence of progressive pulmonary granulomatosis even among cases available for study, much less in the group lost for follow-up purposes.—*Stanley M. Rogoff, M.D.*

Enterline, Philip. Group chest x-ray examinations and the tuberculosis death rate. *Pub. Health Rep.*, Aug., 1952, *67*, 762–766.

Mass x-ray projects are defined as "any group x-ray examinations, including contacts regardless of the size of films, taken for the purpose of detecting pulmonary pathology, but not for the identification of pathology or determination of activity."

In 1946 about 6 million examinations were made in the United States for the detection of chest disease excluding chest roentgenograms by the Armed Forces and the Veterans Administration.

During 1950 it is indicated that about 15 million examinations were made.

From 1947 to 1950 the tuberculosis death rate dropped from 33.5 to 22.2 per cent, a decline of 34 per cent.

Factors contributing to this favorable experience may be betterment in housing and general living conditions, improvement in case finding and advances in therapy.

There appears to be a relationship between the intensity of the case finding activities and the decline in the death rate as demonstrated by the author in nine geographic regions tabulated in the article.

However, with intensified case finding activity there are also present improved treatment facilities, standards of living and over-all improved health department activities, and the author correctly evaluates the correlation of all of these important factors.—*W. W. Maver, M.D.*

McGrath, Edward, J., Gall, Edward A., and Kessler, Dale P. Bronchiogenic carcinoma, a product of multiple sites of origin. *J. Thoracic Surg.*, Sept., 1952, *24*, 271–283.

A formidable amount of evidence has been gathered in recent years indicating the multicentric origin of many tumors. Except for the diffuse alveolar cell tumors, little attention has been directed to this phenomenon in bronchiogenic carcinoma.

The authors took large sections from 87 lungs containing bronchiogenic carcinoma. In 5 of these there were 2 grossly independent primary lesions. In 19 others there were found on microscopic examination one or more independent carcinomas which lacked continuity with the main carcinoma. In 46 cases the major presenting tumor contained 2 to 5 different histopathologic patterns arranged in such a way that the authors believe they represented initial independence and ultimate fusion of neighboring neoplasms.

The majority (63 per cent) of the specimens examined exhibited one or more of the features enumerated.

It is believed that the aggregate of these observations lends weight to the hypothesis that bronchiogenic carcinoma may often arise from more than one site and that frequently clinical recognition does not occur until coalescence of independent growths has been completed.—*Thomas Stansbury, M.D.*

Whitesell, Frank B., Jr., and White, William J. Congenital cystic disease of the lung in the newborn; report of a successful left lower lobectomy in a seven-day-old infant. *Ann. Surg.*, Aug., 1952, *136*, 299–304.

Cystic disease of the lung is a rare condition but one which may be a surgical emergency in the newborn. Respiratory distress and cyanosis are the clinical findings encountered. They may appear immediately post-partum or may not be evident for several hours or days.

The cysts are of two general types: those containing fluid and those containing air. With the fluid filled cysts the degree of respiratory distress remains static and is directly proportional to the size of the cysts at birth. Those containing air usually have a bronchial communication, often allowing air to flow in but trapping it on expiration. Air containing cysts may thus rapidly increase in size. Because the mediastinum is flaccid, the expanding cyst is limited only by the thoracic cage so that its compression and distortion of the vascular and pulmonary structures may become sufficient to be fatal. If not, with delay, infection of the cyst and its contents is a further hazard. In addition, the frequent limitations of the disease to a single lobe and the technical ease of pulmonary surgery in the newborn makes early diagnosis of great importance.

Recognition of these lesions is based upon the roentgen findings of an air containing lesion displacing the heart and mediastinum to the opposite side and depressing the diaphragm. It must be differentiated from agenesis of the lung, which produces an ipsilateral shift. Multilocular air containing cysts must be distinguished from a congenital diaphragmatic hernia in that its loculations may be mistaken for loops of bowel. Studies by means of lipiodol by mouth or barium enema may be necessary.

A case of a seven day old infant, treated by lobectomy, is presented.—*John F. Weigen, M.D.*

Johnson, E. K., and Mangiardi, J. L. Diaphragmatic hernia in the newborn. *A.M.A. Am. J. Dis. Child.*, Oct., 1952, *84*, 436–438.

A case of paraesophageal hiatus hernia is reported in this paper. The authors state that the condition is a congenital defect that is usually found only in adults. Its incidence in general is fairly low, while in infants or the newborn it is extremely low. Only 4 cases have been reported of its occurrence in infants and none of its occurrence in the newborn. The early correction of the herniation through an abdominal approach seemed justified in their case and a successful repair operation was performed on the second day of life. A roentgenogram of the chest showed a gas-filled viscus in the mediastinum, projecting to the right and dorsally from the heart. The diagnosis was confirmed by injection of iodized oil in the esophagus.—*Ralph S. Bromer, M.D.*

Beck, William C., and Motsay, Dominic S. Eventration of the diaphragm. *A.M.A. Arch. Surg.*, Oct., 1952, *65*, 557–563.

Eventration of the diaphragm is due to a congenital aplasia, either partial or complete, which permits the protrusion of the abdominal viscera into the chest.

In most instances partial eventration produces no symptoms. Partial eventration of the right diaphragm is fairly frequent. Chest roentgenograms show an upward bulge of part of the diaphragm which can erroneously be considered to be caused by an accessory lobe of the liver or a tumor mass in the liver.

In infants and children complete eventration may produce serious respiratory distress with dyspnea and cyanosis.

The diagnosis of complete eventration is revealed on a routine chest roentgenogram. A marked elevation of the right or left side of the diaphragm can be seen. Fluoroscopic examination may be necessary for differential diagnosis. One may see a small amount of normal motion, minor paradoxical motion, or no movement.

The most important differential diagnosis is from congenital atelectasis. In eventration the thoracic structures are pushed toward the normal side, while in atelectasis they are drawn toward the abnormal side.

The most difficult differential diagnosis is between this condition and congenital diaphragmatic hernia. In some cases this may not be possible but it is desirable from a surgical repair point of view to be able to differentiate between an eventration and a hernia without a true sac.

Surgical correction can be carried out in most infants without difficulty.—*Wallace M. Roy, M.D.*

Ebbs, J. H., and McGarry, H. H. Congenital hernia of the diaphragm. *Canad. M. A. J.*, Aug., 1952, *67*, 115–117.

The reason for infrequent incidence of congenital type diaphragmatic herniation in adult patients is that 75 per cent of children suffering from this disorder die before they are one year old. The following surgical statistics have been recorded.

Orr and Neff (1936) 17 patients with mortality of 47 per cent.

Ladd and Gross (1940) 16 patients with mortality of 44 per cent.

Thomson (1950) 12 patients with mortality of 16 per cent.

The authors add a case report of a patient three months of age with a story of wheezy respirations and having "failed to thrive." Roentgen examination revealed displacement of the mediastinal structures toward the right. Intestinal loops were observed in the left chest and partial collapse of the left lung was noted. Immediate surgical repair of the defect in the left dome of the diaphragm was followed by return to normal health and growth.

A plea is made for early diagnosis and early surgical repair—preferably in the neonatal period.—*John W. Turner, M.D.*

DeCarlo, John, Jr., Tramer, Arnold, and Startzman, Henry H., Jr. Iodized oil aspiration in the newborn. *A.M.A. Am. J. Dis. Child.*, Oct., 1952, *84*, 442–445.

During fluoroscopy of the esophagus in newborn infants, with the use of iodized oil, the authors frequently observed the presence of the oil in the trachea and the bronchial tree. One hundred healthy newborn infants were studied. They varied in ages from twelve to twenty-four hours and were examined in the supine position with a standard fluoroscopic-roentgenographic unit. Those studied within the first twelve prenatal hours received no feeding of any kind. After a preliminary fluoroscopic survey of the chest and abdomen, they were fed varying amounts of chloriodized oil (iodochlorol), warmed to body temperature, a standard feeding bottle and nipple being used. The act of swallowing was carefully observed and the opaque medium was followed to the stomach. Spot films were made of all unusual findings. Patients who aspirated radiopaque material were re-examined at successive twenty-four hour periods until aspiration ceased. Infants who aspirated chloriodized oil were restudied, barium sulfate and water mixtures being fed under the same conditions.

Although each chloriodized oil bolus traversed the hypopharynx and entered the esophagus without delay, a small amount remained in the laryngeal area until successive acts of deglutition cleared the larynx of the opaque material. In 13 per cent of their cases the bolus divided at the larynx, with the greater part of the bolus entering the esophagus and the remainder passing into the trachea. This finding was infrequently noted on the first swallow but was

usually noted on the third or fourth bolus of the medium. The amount of material aspirated by the abnormal group seemed to bear a direct relationship to the vigor with which the feeding was taken. When a barium mixture was substituted for the oil, there were no instances of aspiration.

The authors believe that the aspiration probably results from neuromuscular immaturity and from the low surface tension of the iodized oil, and that iodized oil is of limited value in the early roentgenographic diagnosis of sublaryngeal tracheoesophageal fistulae.—*Ralph S. Bromer, M.D.*

Gershon-Cohen, J., and Ingleby, Helen. Secretory disease and plasma cell mastitis in the female breast. *Surg., Gynec. & Obst.*, Oct., 1952, *95*, 497–504.

Secretory disease represents an abortive attempt at secretion on the part of a nonpuerperal breast and is not an inflammation, although inflammation may be a sequel to the disease.

In their study of 24 cases of secretory disease, the authors discovered that the condition is usually bilateral and that it is more common than is generally thought. In the majority of the cases which are due to chemical irritation, inflammation of the type known as plasma cell mastitis supervenes. In general, it is the plasma cell mastitis rather than the secretory disease which is responsible for the symptoms, although nipple discharge is common to both.

Secretory disease and plasma cell mastitis are not precancerous conditions. Treatment is optional but if surgery is undertaken the whole of the diseased area should be excised. Otherwise, complications such as discharging sinuses may follow.

The diagnosis may be suspected from the history and physical examination and is confirmed in most cases by the characteristic roentgen findings and by examination of a smear from the nipple discharge.

Roentgenology. The lesion is most easily identified when it is situated in the nipple area. The ducts leading from the nipple are visible because they are distended. Many or all may be involved; it is rare to find only one duct affected. Their course toward the base of the gland is tortuous and they end in club-shaped dilatations. A sizable focus of plasma cell mastitis is seen in the roentgenogram as a homogeneous, coarsely tentacled density, easily visible in older breasts where there is much perifocal fat. The margins of these masses extend in flame-like projections along the line of the trabeculae. In the younger group of patients, the most striking roentgen finding is the uniform ground glass appearance of the affected area.

Pathology. If nipple discharge is present, the diagnosis can almost always be made from the smear. Unless degeneration of the duct contents is far advanced, colostrum cells are recognizable. Besides colostrum, inflammatory cells of various kinds are apt to be present and would be expected where plasma cell mastitis is a complication. In plasma cell mastitis, fatty acid crystals may be found and even giant cells and "rosettes." The gross appearance of secretory disease is that of worm-like casts exuding from the dilated ducts. In uncomplicated cases the inspissated material is white or yellowish. When it shows red, green or brown discoloration, there is likely to be plasma cell mastitis.

Discussion. Secretory disease, while allied to other forms of mammary dysplasia, deserves to be classified as a distinct entity. It is a much milder disorder than its rather bizarre appearance would suggest, and it is only important as the precursor of plasma cell mastitis. Since the essential feature is differentiation of epithelial cells, followed by degeneration and shedding, carcinoma does not arise in the affected ducts.—*Mary Frances Vastine, M.D.*

Schreiber, Friedrich-Karl. Beitrag zur röntgenologischen Diagnostik der Lungenspitzen. (On the roentgenological visualization of the lung apices.) Tuberkulosearzt, Oct., 1952, *6*, 585–591.

The author considers laminagraphy the most reliable method to eliminate the superimposition of the clavicles and upper ribs and demonstrate clearly the lung apices. When laminagraphy is not available roentgenograms of the apices taken with angulation of the tube by 45° in the caudal direction are recommended. The technique is the same as in the ordinary posteroanterior chest films, except that the focus film distance is only 3 feet and the central ray passes through the jugular fossa. Elevation of the patient's arms above the head sometimes improves the result. The same milliampere and kilovoltage settings as for an ordinary chest film are used; the increased thickness due to obliquity of the radiation compensates for the shorter distance.

The author illustrates the usefulness of this projection by 17 roentgenographic reproductions.—*Karol E. Matzinger, M.D.*

Abdomen

Olivier, Cl., Faurel, and Joffre. Radiodiagnostic de la dilatation aiguë spontanée de l'estomac. (Roentgen diagnosis of acute spontaneous dilatation of the stomach.) *Presse méd.*, Oct. 1, 1952, *60*, 1284–1285.

This diagnosis ordinarily rests on the contrast between clinical signs of intestinal obstruction with rapid and marked abdominal distention as against the roentgenologic finding of a general dense abdomen with a large left subphrenic fluid level.

The diagnosis must also be considered when, in the presence of clinical signs of peritonitis, there is roentgen evidence of a very large gastric fluid level without pneumoperitoneum.

A barium enema may show compression and displacement caudad of the transverse colon providing that spasm does not prevent its filling.—*Marcy L. Sussman, M.D.*

Curtis, William S. Chronic gastritis; a review of the literature. *Radiology*, Sept., 1952, *59*, 317–323.

Chronic gastritis is a controversial subject with differences of opinion among and between the various specialists.

The history on the subject of gastritis is traced from the Period of Ascendancy, 1728–1838; to the period of the Wane of Interest, 1838–1922; and through the Reawakening, 1922–1951. Since 1951 there has been a re-evaluation of, "What is gastritis and what is a normal stomach?"

The difficulties of evaluation are discussed. The gastroscopists and pathologists agree fairly well on the classification and findings in (1) superficial gastritis, (2) hypertrophic gastritis, and (3) atrophic gastritis. Antral gastritis, which is the hypertrophic type, is considered by the radiologists, pathologists and surgeons to be the most common form of hypertrophic gastritis, whereas the gastroscopist, who cannot well visualize this portion of the stomach, says it is uncommon.

The etiological factors considered are: (1) irritants, (2) hematogenous agents, (3) mechanical factors, (4) nervous mechanism, and (5) allergy.

The pathology depends upon the duration and stage of inflammation. Probably all forms represent different types of response to the same factors.

The symptoms of atrophic gastritis are unrelated to meals and alkali and produce no pain. Hypertrophic gastritis simulates prepyloric or duodenal ulcer symptoms.

Acute or chronic gastritis may be a precursor of ulcer. Chronic hypertrophic gastritis does not seem to be a precursor of cancer, but it is questionable whether atrophic gastritis may be.

It is granted that roentgen examination is of little value in diagnosing atrophic gastritis, but is of considerable aid in diagnosing hypertrophic gastritis.

Although the distinction is often difficult, it is extremely important that radiologists learn to differentiate hypertrophic gastritis from ulcer, and particularly from cancer.—*Everett E. Seedorf, M.D.*

Berg, H. M. Antral gastritis. *Radiology*, Sept., 1952, *59*, 324–335.

In a consecutive series of 5,520 roentgen examinations of the stomach, over a thirty month period, the diagnosis of antral gastritis was made 139 times. All cases in the series were of the type usually classified as hypertrophic antral gastritis by the radiologist.

Roentgen changes seen in antral gastritis are listed as follows:

1. Hypersecretion.
2. Pylorospasm, antral spasm, and narrowing of antral region.
3. Shortening of antral region due to contraction of longitudinal muscles.
4. Gastric folds usually enlarged, sometimes with polypoid appearance. (Some cases are seen, however, in which the mucosa is smooth, as it appears in scirrhous carcinoma.)
5. Stiffness of folds in antral region due to infiltration of submucosa. (Folds do not flatten out on pressure of the examiner's hand and do not thin out with peristalsis.)
6. Irregularity of peristalsis in antral region.
7. Prolapse of gastric mucosa into duodenum.
8. Hypertrophy of pyloric musculature in some cases.
9. Delay in emptying time of stomach due to antral spasm and pylorospasm.
10. Small ulcer craters sometimes detectable in involved area.

It is difficult to differentiate antral gastritis from other lesions that occur in this area, such as prepyloric ulcer, hypertrophy of the pylorus, and carcinoma. The differential diagnosis of these lesions is discussed.

Two of the 59 cases of antral gastritis without other demonstrable lesions in the stomach or duodenum came to surgery and the diagnosis was confirmed. The other 57 cases have all been followed for more than two years. No malignant change was found in any of these. In 80 cases the antral gastritis was associated with duodenal ulcer, duodenitis, gastric ulcer, or gastroenterostomy.—*Arno W. Sommer, M.D.*

Stampfli, Wendell P. Roentgenologic exploration of gastric rugae with reference to the diagnosis of gastritis. *Radiology*, Sept., 1952, *59*, 336–348.

A method for studying gastric rugae is presented. While in the erect position the patient swallows 1 ounce of a fairly thick barium mixture. Palpation and examination in the erect, and the recumbent prone and supine positions, distributes the barium to coat the entire gastric lining. This permits visualization of mucosal detail. Double contrast study of the stomach, antrum and duodenum is made possible by the air normally present. If swallowed air is not sufficient, a carbonated barium suspension will usually produce a satisfactory increase in the amount of air and better visualization.

After this preliminary examination the patient is watched as he swallows 7 ounces of a thinner mixture of barium. Palpation and inspection are carried out in the usual way and spot roentgenograms made with or without pressure. This part of the study is again done with the patient erect and recumbent.

The method enables the radiologist to visualize, and to record on film, mucosal detail throughout the stomach, and permits identification of changes due to gastritis. In gastritis the rugosity of the stomach usually is more pronounced than normal. The folds are often more tortuous and redundant. Prolapse of the thickened mucosa through the pylorus is common. Extreme thickening and rigidity of the stomach lining are sometimes seen without rugosity. Hypersecretion is observed frequently. Peristaltic contractions are apt to be sluggish and ineffective. Motility of the gastric mucosa may be limited. Antral narrowing is one of the most common and important signs.—*Arno W. Sommer, M.D.*

Ingersoll, Charles F. Speculations concerning the probable evolution of chronic gastritis. *Radiology*, Sept., 1952, *59*, 349–357.

The author believes that chronic gastritis passes through phases of increasing and progressively irreversible pathologic changes in the stomach. Manifestations of these changes in such order may be indicated by: prolapsing gastric mucosa, antral gastritis, generalized gastritis with a prominent antral phase, and finally, antral and pyloric fibrosis and hypertrophy (frequently called benign hypertrophy of the pylorus in adults). It is granted that this concept conveniently ignores isolated hypertrophy other than antral, but the number of such cases is regarded as insignificant.

It is believed that gastritis of the antral mucosa stiffens it sufficiently so that it is propelled through the pylorus, thereby producing prolapse. As the extent of the gastritis increases, the antral narrowing phase is noted. After years of antral gastritis, hypertrophy of the musculature develops together with fibrosis due to the chronic inflammation, thereby producing adult hypertrophy of the pylorus.—*Martin B. Goodwin, M.D.*

McGlone, Frank B. Clinical aspects of gastritis and the gastroscopic findings. *Radiology*, Sept., 1952, *59*, 358–366.

Of a group of 2,400 patients who were studied gastroscopically, the author selected 10 cases for presentation. These particular cases were chosen to illustrate the more important clinical problems associated with gastritis.

The cases were divided into those with antral gastritis, those with diffuse involvement and those with lesions of the cardia. Patients with antral defects or those with diffuse lesions are frequently difficult to diagnose by means of the gastroscope. Roentgen examination and other methods help in many cases, but surgical exploration is necessary in some instances to differentiate gastritis from malignancy.

Lesions of the cardia are usually due to enlarged mucosal folds or neoplasm, and therefore a diagnosis of localized hypertrophic gastritis in the cardia should be regarded with caution.

The author concludes that although gastritis is usually diagnosed by gastroscopy, some cases will require all the combined diagnostic facilities of the physician to arrive at a final decision in regard to therapy.—*McClure Wilson, M.D.*

Kennedy, Thomas J. Difficulties in the differential diagnosis of gastric carcinoma and gastritis; surgical consideration. *Radiology*, Sept., 1952, *59*, 367–370.

Even though the roentgenological and gastroscopic modalities have a fairly high degree of accuracy, it is impossible in many cases to differentiate clinically between a benign and malignant gastric process.

In view of the above fact and inasmuch as gastritis has been postulated as a probable precursor of carcinoma of the stomach, the author believes that a diagnosis of either condition calls for surgical exploration.—*McClure Wilson, M.D.*

Larson, L. W., and Kling, R. R. Pathology of gastritis. *Radiology*, Sept., 1952, *59*, 371–376.

The authors discuss the gross and histopathology of gastritis. In reviewing the anatomy of the gastric mucosa it is stated that the presence of moderate to many lymphocytes in the gastric mucosa of adults is now considered normal.

A classification of gastritis proposed by Benedict and Mallory, which is dependent upon the degree and type of lymphocytic infiltration, is presented.

The presence of intestinal type of epithelium and glands in the gastric mucosa is considered indicative of gastritis.

The radiologist is concerned with the size and character of the gastric rugae. The rugae are altered by fibrous tissue infiltration into the muscularis mucosae and submucosa which affects the tone of these structures.

The manner in which a given case of gastritis is classified will depend upon whether hypertrophic, atrophic, or exudative changes predominate. Mixed types may occur.

Seven figures of histological variants are presented.—*Everett E. Seedorf, M.D.*

Dublin, William B. Problem of pathogenesis of chronic hypertrophic gastritis. *Radiology*, Sept., 1952, *59*, 377–383.

Many theories, equally unsupported, have been advanced as to the probable etiology of chronic hypertrophic gastritis. Ingested irritants such as alcohol and condiments, bacteria and their toxins, allergens, virus infections, drugs, endocrine disorders, and neurogenic factors have all been mentioned.

The author proposes that the neurogenic factor be strongly considered and he suggests the following pathogenesis: Excessive neurogenic activity produces excessive gastric secretion together with hypercontractility. The latter leads to ischemia of gastric tissue, making it more vulnerable to excess acid. In addition, the mucinous secretion may be poorly balanced against the acid and pepsin. The hypercontractility can produce gross structural deformity, enlarged rugae, and mucosal irregularity, these abnormalities disappearing when the specimen reaches the surgical pathologist's hands. Continued hyperirritability and hypersecretion, however, will produce pathologic changes, the earliest of which are edema and congestion usually with lymphocytic infiltration. Chronic repeated insult of this type eventually results in the late changes of gastritis, fibrosis and hypertrophy of the musculature.

Prolapse of the gastric mucosa is believed to occur after rugae stiffened by infiltrations are subjected to hyperperistalsis. Duodenal ulcer, duodenitis, and/or gastric ulcer are often associated with chronic gastritis.—*Martin B. Goodwin, M.D.*

ALBARRACÍN, A. El síndrome del "dumping" gástrico (The syndrome of gastric "dumping.") *Bol. Inst. Pat. Méd.*, Sept., 1952, *7*, 161–165.

Excluding recurrences, 11 per cent of patients undergoing gastrectomy have postprandial symptoms, although only about 2 per cent are intense. Symptoms may appear following eating quite soon (authentic dumping) or may occur at a later time (postgastrectomy hypoglycemia.)

In the author's experience, an absolute rhythm of discomfort occurring soon or late after eating was not noted. With meals high in carbohydrate value there was an abrupt rise and fall in the blood glucose curve, leading to early postprandial symptoms. Hypertonic solutions of various salts provoked symptoms. With the accelerated gastric emptying following partial gastrectomy, there was enhanced intestinal peristalsis. In addition, intestinal distention, jejunitis and autonomic alterations were found to change the clinical picture.

Fundamental therapy should be directed against osmotic alteration. To ingest isotonic aliments would require large volumes for sufficient nutrition. By giving solid food, followed a half hour later with liquid, the patient may be more comfortable. If neurovegetative symptoms are present, administration of atropine or luminal is indicated.

Four cases are presented with this type of approach in mind with prompt cessation of symptoms. —*Charles M. Nice, M.D.*

HENLEY, F. A. Gastrectomy with replacement; a preliminary communication. *Brit. J. Surg.*, Sept., 1952, *40*, 118–128.

A preliminary report is given of the operation of replacement of part or the whole of the stomach by a segment of jejunum. This operation has been performed 35 times up to March, 1952, without mortality. Complications have been 6 in all, 2 necessitating further surgery, but the remaining 4 responded to medical treatment.

Postoperatively the roentgenographic appearance is as follows: Barium enters the gastric remnant and appears to be milked by the peristaltic action of the jejunal graft into the duodenum. The average emptying time of the stomach and graft is two hours. So far, neither the gastric stump nor the jejunal graft have undergone dilatation and the jejunal graft has maintained its normal tone.—*James J. McCort, M.D.*

MAGGI, A. L. C., MEEROFF, M., and SEGAL, J. E. Radiología de la úlcera gastroduodenal; estudio estadístico de 100 casos. (Roentgenology of gastroduodenal ulcer; statistical study of 100 cases.) *Prensa méd. argent.*, Oct., 24, 1952, *39*, 2565–2574.

Statistics are presented on 100 consecutive male patients with gastroduodenal ulcer. Topographically there were 21 gastric ulcers, 75 duodenal ulcers, 3 double ulcers (gastric and duodenal), and 1 patient was designated simply as having peptic ulcer.

Of the 21 gastric ulcers, 17 were on the lesser curvature, 1 in the antrum, 2 in the prepyloric region and 1 in the pylorus. Of the 17 ulcers on the lesser curvature, 2 were on the superior third, 9 on the middle third, 4 at the junction between the middle and lower thirds, and 2 on the inferior third. Four of the cases presented cicatricial contraction. No giant niches were seen. Convergence of folds was observed in 4 patients, retraction of the zone of implantation in 3, segmentary rectitude of the affected zone in 3, opposing spastic incisura in 1, and a niche "en botón de camisa" ("collar-button niche") was noted in 1 patient. Of 17 verified niches, there was a centric, permeable pylorus without spasm in 16 patients. In 1 the pylorus was centric but there was spasm of the pylorus. Three had megabulbus, 1 had a distended, paretic bulb and 1 presented irritability with rapid empyting of the bulb.

Of the 75 duodenal ulcers, 30 had a visible niche, 28 had indirect signs, and 17 presented deformity of chronic duodenal ulcer. Of the 30 patients with a visible niche, 18 had a niche on the lesser curvature, 9 on the greater curvature and 3 on the posterior wall. Indirect signs in the 28 patients without visible niche included the following: gross edema, enlarged converging folds in 15 patients; gross edema, enlarged converging folds and mesobulbar spasm in 6; irregular image with rapid emptying in 7 patients. Signs coexisting with duodenal niche included mesobulbar spasm in 14 patients, dilatation of duodenal recess in 5, diverticular type of dilatation in 2, bulbar

retraction in 4, and absence of alteration in 5 patients. Megabulbus was present in 7 of the 30 patients.

Other statistics are quoted and compared with the series of other authors.—*Charles M. Nice, M.D.*

Evans, John A. Gastroileostomy. *Radiology*, Oct., 1952, *59*, 573–577.

Gastroileostomy is a comparatively rare surgical error. While the history and symptoms in this condition may be suggestive, there is no constant, typical or pathognomonic clinical picture. Roentgen findings, however, are usually specific.

Symptoms include weight loss, diarrhea, vomiting, hemorrhage, and severe pain if marginal or ileal ulceration is present. The roentgen findings depend on the type of surgical procedure. With gastric resection, barium passes rapidly into the ileal anastomosis and quickly appears in the cecum. In gastroileostomy without gastric resection the barium leaves the stomach both via the pylorus and the gastric stoma and the entire small bowel will be visualized. The diagnostic feature in this instance is the delayed film examination showing refilling of the stomach four or five hours following the barium meal. The barium which leaves the stomach by the pylorus passes through the small bowel until it reaches the ileum and re-enters the stomach through the gastroileostomy.

The author presents a case illustrating this unusual condition.—*H. C. Hamilton, M.D.*

Macpherson, R. A. How the radiologist can assist in the management of the long intestinal tube. *J. Canad. A. Radiologists*, Sept., 1952, *3*, 54–56.

The author discusses the indications as well as the contraindications for the use of an intestinal tube. He prefers the Miller-Abbott tube and gives an outline of his procedure for intubation.

In 180 cases of intestinal obstruction in the Winnipeg General Hospital, intubation was successful in 52 cases. In 27 cases it was used to advantage in preparing the patient for surgery. Twenty-five cases did not require surgery.

It is the author's opinion that the radiologist can best control and supervise the intubation by frequent roentgenographic and fluoroscopic study. In this manner he can share with the internist and surgeon the responsibility of the successful management of cases of intestinal obstruction.—*V. L. Peterson, M.D.*

McKenzie, A. D., Moore, J. R., and Miller, G. Gavin. Complications of gastro-intestinal intubation. *Canad. M.A.J.*, Nov., 1952, *67*, 403–405.

Shortly after the introduction of the procedure of gastrointestinal intubation, various complications of the procedure were reported in the literature. The authors report 4 interesting mishaps associated with the use of the long nasal-gastric tube. The cases include:

1. Inadvertent distention of the balloon with multiple perforations of the jejunum and of the ileum.

2. Perforation of necrotic ileum by the balloon of the Miller-Abbott tube, with production of a pelvic abscess.

3. Loss of the proximal end of the Miller-Abbott tube.

4. Multiple perforations of the small bowel associated with the use of the Cantor tube.

The authors comment briefly on prevention of the complications encountered.—*Moris Horwitz, M.D.*

Glazebrook, A. J., and Welbourn, Richard B. Some observations on the function of the small intestine after gastrectomy. *Brit. J. Surg.*, Sept., 1952, *40*, 111–117.

Observations are described on the relation of the small intestinal motor activity to the efferent loop dumping syndrome and to postgastrectomy steatorrhea.

Inflation of a balloon in the efferent jejunal loop reproduced "dumping" symptoms in 3 out of 23 patients. Kymography showed simultaneous increased motor activity.

Hypertonic solutions, introduced into the jejunum, caused dumping symptoms constantly in 9 cases, and also produced marked activity on the kymogram.

The injection of methonium salts (C_5 and C_6) caused a diminution of the dumping syndrome and profound reduction in jejunal activity and tone.

Hypertonic barium meals produced "dumping" symptoms and marked activity in the jejunum in 5 cases. In 1 case a peristaltic rush caused the head of the meal to reach the hepatic flexure of the colon in five minutes. Methonium salts tended to reduce the activity seen roentgenologically.

The mechanism of efferent loop "dumping" syndrome is discussed and it is concluded that increased motor activity in the jejunum provides the adequate stimulus for the production of symptoms.—*James J. McCort, M.D.*

Gill, A. Morton, and Falle, Elizabeth de C. Recognition of functional disorders of the small intestine. *Lancet*, Aug. 23, 1953, *2*, 356–360.

The authors report 8 cases which had been studied because of abdominal pain, obscure in origin and resistant to therapy, and which had roentgenologic evidence of abnormality of the ileum. Functional disorders of the colon are well recognized but similar

conditions occurring in the small intestine are not as well known.

In this series the patients were females who had bizarre abdominal complaints. Nausea, distention, abdominal pain and disordered bowel function with either constipation or diarrhea were present in varying degrees in all cases. Operation failed to reveal organic disease of either the large or small intestine. Reproductions of roentgenograms and tracings of the abnormal areas of small intestine are presented in the paper and are reported to demonstrate irritable loops of ileum, localized ileal spasm, clumping and segmentation, and abnormality of the mucous membrane pattern of the ileum.

The authors believe that these roentgenographic changes should be recognized in cases of intestinal dysfunction due to functional disorders of the small bowel, the ileal syndrome.—*Gerald Lavner, M.D.*

Hemley, Samuel D. Incidental diagnosis of carcinoma of the right colon during cholecystography. *Am. J. Digest. Dis.*, Sept., 1952, 19, 295–298.

Gas within the bowel which results from the administration of priodax may under certain conditions be of help in the diagnosis of carcinoma of the right colon. The author cites the case of a sixty-eight year old female whose roentgenograms revealed cholelithiasis and a biliary fistula. At operation she was also found to have an annular lesion 3 to 4 cm. in diameter at the junction of the middle and proximal thirds of the transverse colon. When the preoperative gallbladder films were re-examined a hiatus in the visualized portion of the right side of the transverse colon was noted in the exact site of the neoplasm. Two other cases with known carcinomas of the right colon also showed similar filling defects in the gas filled colon when cholecystography was performed.

It is concluded that more attention should be paid to possible defects shown in the colon during cholecystography, and if there is any suspicion of such, further investigation should be instituted.—*Arthur E. Childe, M.D.*

White, Matthew, and Dennison, Wallace M. Irreducible intussusception in infants. *Brit. J. Surg.*, Sept., 1952, *40*, 137–140.

A series of 16 cases of irreducible intussusception treated by simple anastomosis is presented. There was no constant relationship between the duration of the condition and irreducibility.

The irreducibility is said to be essentially a vascular catastrophe which may occur at an early stage. The manner in which the intussusception becomes irreducible is believed to be as follows: When the normal process of boliteration of the mesentery has taken place, the cecum is fixed in position and the ileum is forced through the ileocecal valve and advances at its own expense. The mesentery of the ileum, however, soon becomes constricted as it enters the intussusception and the blood supply is obstructed, with consequent edema and congestion. The peritoneal surfaces of the inner and middle layers which are in contact become adherent and further advance is checked. Reduction becomes impossible. The vascular obstruction eventually leads to gangrene of the bowel but usually only the inner layer is affected and the ensheathing layer remains viable. Death occurs from intestinal obstruction and toxemia.

The authors have never seen death from peritonitis in an unreduced intussusception. The return of the irreducible mass into the peritoneal cavity does not therefore violate surgical principles. When there is any doubt about the viability of the bowel the mass is exteriorized and resection carried out as soon as the condition of the patient permits.—*James J. McCort, M.D.*

Dean, David, Lea, Volvulus of the cecum; with report of two cases. *Ann. Surg.*, 1952, *136*, 319–325.

Volvulus throughout the gastrointestinal tract constitutes about 10 per cent of the causes of intestinal obstruction. It occurs most frequently in the sigmoid flexure, less frequently in the cecum and small intestine, and rarely in the stomach or transverse colon.

Intestinal obstruction due to volvulus is accompanied by a high mortality rate. Even in those cases treated surgically the mortality rate is found to be between 50 and 60 per cent. If gangrene is present the mortality rises to 80 per cent; 100 per cent mortality occurs in the cases not operated upon.

Volvulus of the cecum occurs as a result of an abnormal mobility of the cecum and ascending colon. Incomplete fixation after rotation and descent in the development of the colon predisposes to it. Pathological changes rarely begin before 180° rotation. In nearly all cases the venous circulation is interfered with and there is marked passive congestion. This may progress to infarction of the wall, gangrene, and perforation. In incomplete obstruction or when the obstruction develops gradually, both gas and fluid are found in large amounts in the twisted loop. If the obstruction is complete, the fluid will usually exceed the gas because the closed loop is cut off from the oral source of swallowed air.

The signs and symptoms are those of a complete low intestinal obstruction with sudden onset of severe colicky pains, usually not localized. Abdominal distention follows, which is not relieved by gastric suction or enemas. The previous history of similar transient attacks of shorter duration and milder course may be obtained.

The diagnosis is made by means of the roentgen examination of the abdomen by the following find-

ings: (1) a dilated cecum in an abnormal position (90 per cent in the left upper quadrant); (2) loops of distended small bowel on the right of the cecum; (3) usually a single fluid level in the upright film. Confirmation is obtained by means of a barium enema by demonstrating a cone-shaped obstruction of the ascending colon, with spiral mucosal folds, and with the ileocecal valve often to the right of the distended viscus.

Earlier diagnosis and treatment is necessary if the mortality rate of 50 per cent is to be reduced. Dependence upon conservative methods such as gastric suction, enemas, etc., will only invite disaster and increase the mortality rate.—*John F. Weigen, M.D.*

Isaacs, Ivan. Intraperitoneal escape of barium enema fluid; perforation of sigmoid colon. *J.A.M.A.*, Oct. 18, 1952, *150*, 645–646.

The author describes 2 instances of perforation of the sigmoid colon in the course of barium enema examination. The predisposing causes are given as diverticulitis, chronic ulcerative colitis, carcinoma, and recent proctoscopic examination.

In the first case immediate laparotomy disclosed free gas, milky fluid, and black exudate. The perforation occurred through a carcinoma.

In the second case it is believed that the perforation was caused by recent sigmoidoscopy. Laparotomy in this case disclosed generalized peritonitis with barium in the peritoneal cavity. The perforation in the sigmoid was associated with no other visible or palpable abnormality.

The author stresses the roentgenological criteria upon which a diagnosis of intraperitoneal escape of barium can be made. One of the characteristic findings is a linear streak of barium seen lateral to the sigmoid and parallel to the descending colon. This finding was noted in both cases reported. The straight dense line along the left lateral margin of the descending colon is attributed to delimitation of the free barium by the parietal peritoneum.—*C. L. Hinkel, M.D.*

Gagliardi, Raymond A., and Gelbach, Philip D. The "reformed gallbladder": clinical and roentgen aspects. *Am. J. Digest. Dis.*, Sept., 1952, *19*, 298–301.

Poor results follow cholecystectomy in possibly 30 to 40 per cent of patients if cholelithiasis is not present. The possible explanation for the recurrence of symptoms in these patients can usually not be expected from the various roentgen techniques now available. However, the condition sometimes called the "new gallbladder" or "reformed gallbladder," with stone or inflammation of a dilated cystic duct remnant is a situation in which roentgen methods may be employed, often with definitive results.

A number of cases of this syndrome have been reported and the authors add another. In this case comma shaped calcification was noted in the right upper quadrant three years after cholecystectomy. This was proved to be extrarenal, and operation showed that it consisted of one stone in the fundus of a "reformed gallbladder" and another smaller one in the duct.

A diseased dilated cystic duct remnant should be considered especially when the recurrent symptoms are identical with those experienced preoperatively. Diagnosis is somewhat facilitated if an opaque calculus can be demonstrated in the gallbladder region. In any event cholecystography should be done regardless of the past history of gallbladder removal.—*Arthur E. Childe, M.D.*

Swenson, Orvar, and Fisher, John H. Utilization of cholangiogram during exploration for biliary atresia. *New England J. Med.*, Aug. 14, 1952, *247*, 247–248.

The authors of this concise and informative article point out that the results of surgical exploration of infants (usually 4–8 weeks of age) with biliary atresia have been discouraging, for only a small percentage of these infants have proved to have a mechanical block that could be relieved surgically.

They state that the surgeon usually explores the regions of the common bile duct, hepatic duct and porta hepatis with the hope of finding a dilated duct system proximal to an atresia, yet in most cases no dilated hepatic or common duct is found. Instead, these structures are of small caliber and are assumed to be atretic. Since some of these babies, despite a hopeless surgical prediction, completely lost their icterus and regained good health, the operative routine was simplified to reduce the danger of injury to the small structures, which, in the patients who made recoveries, were evidently capable of becoming dilated and functionally adequate. The first innovation in technique was to insert a needle into the gallbladder and inject methylene blue solution before any dissection was done. Blue dye in the duodenum proved patency of the cystic duct and common bile duct. The gastrohepatic ligament was then explored to rule out an atresia of the hepatic ducts. Again, a number of these patients recovered completely. Recently, they found that outlining the hepatic system with diodrast and obtaining roentgenograms taken with portable apparatus in the operating room simplified the procedure still further and eliminated the possibility of damage to a minute but patent duct system.

The technique for the on-the-table cholangiography is described in detail.

The authors have investigated 4 cases in this manner. In 3, a patent biliary system was demonstrated by cholangiograms. In 1 case, no duct system was found.

They state that the cholangiographic method has two advantages: the small surgical procedure pro-

duces a minimal risk to the patient, and there is no chance of traumatizing the small ducts.

In conclusion, it is postulated that some of the cases, diagnosed as biliary atresia because of inability to demonstrate a patent extrahepatic biliary tree, fall into the group of mucoviscidosis. The jaundice is thought to be due to thick, tenacious biliary secretions inspissated in the bile ducts, causing mechanical obstruction.—*Ralph F. Niehaus, M.D.*

Gynecology and Obstetrics

Crichton, Derk. The accuracy of x-ray cephalometry *in utero*. *Proc. Roy. Soc. Med.*, Aug., 1952, *45*, 535–538.

The primary concern of this paper is the measurement of the size of the fetal head with particular reference to the diagnosis of cephalopelvic disproportion. The presenting area of the head is reduced by as much as 25 per cent by flexion of the head and molding. For this reason cephalopelvimetry, even if completely accurate, does not replace clinical evaluation of the progress of labor. The determination of cranial circumference allows for greater precision than the estimation of the biparietal diameter. The allowable error is considerably less in estimation of the biparietal diameter than an estimation of the cranial circumference. The position of the fetal skeleton in the maternal pelvis frequently makes it impractical to measure the biparietal diameter. Of 214 cases examined in this series, the biparietal diameter could not be measured in 33 per cent. The inability to obtain this diameter occurs more frequently when it is most desired.

After thirty-seven weeks of gestation, the biparietal diameter increases approximately 1 mm. per week. If pelvimetry is performed before term, this rule of thumb assists in estimation of head size at term.

Fetal heads vary in their relative diameters, and it is the conclusion of the author that the determination of the biparietal diameter, based upon measurement of another cranial diameter, is fraught with considerable danger of inaccuracy. The inaccuracy of head size prediction plus the relative changes produced by flexion and molding makes it impossible to place absolute reliance upon the roentgenological diagnosis of disproportion.—*Donald R. Bernhardt, M.D.*

MacGregor, W. G., and Oliver, R. Hysterosalpingography; to screen or not to screen. *Lancet*, Sept. 20, 1952, *2*, 563–564.

The authors carried out measurements on 4 patients and 1 cadaver in an attempt to estimate as accurately as possible the radiation dose received by the patient during hysterosalpingography in the combined screening and roentgenographic technique. The average examination involves one or two minutes' screening and two roentgenograms taken at 40–50 milliampere-seconds each. Under these conditions the largest doses observed were 47 r to the skin and 4 r in the vagina, corresponding to an ovarian dose of about 6 r.

They conclude that long screening is undesirable but that this level of dosage does not seem to rule out the use of a screening examination if the physician feels that it is preferable to the direct film technique.—*Arthur E. Childe, M.D.*

Dietz, W. Die Hysterosalpingographie mit wasserlöslichen Kontrastmitteln. (Hysterosalpingography with water soluble contrast media.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Aug., 1952, *77*, 224–229.

The first attempts at visualization of the uterus were made with water soluble contrast media over forty years ago. However, these contrast media caused considerable irritation and their use was soon discontinued and iodized oil was then substituted.

The author states that occasional decomposition products of the oil have caused irritation and he has been using water soluble contrast media for the past three years. Ten to 15 cc. of different mixtures of iodized compounds containing 25 to 50 per cent iodine and having a viscosity of 800 to 1,200 cps are employed and the examination is done under fluoroscopic control.

The advantages of water soluble contrast media are that they are free from danger, the examinations can be done in one sitting and that they mix well with body fluids, permitting visualization of fluid-filled cavities.

The contraindications of this method are the same as with the use of iodized oil.—*Egon G. Wissing, M.D.*

Sánchez, L. Arrieta. Cistografía en el diagnóstico de la placenta previa. (Cystography in the diagnosis of placenta previa.) *Radiología*, Panamá, June, 1952, *2*, 161–166.

Among the different methods which have been utilized for the roentgen diagnosis of placenta previa (amniography, rectal air insufflation, laminagraphy, intravenous placentography, soft tissue differentiation technique, cystography), cystography with radiopaque media has proved its value as the safest and most simple procedure.

The position of the fetal head or its displacement with respect to the median line, widening of the interspace between the fetal head and the upper border of the bladder (over 1 cm.), and appearance of the lateral angles of the bladder are valuable signs in the diagnosis of placenta previa.

The author describes another sign which he considers characteristic. In normal implantation of the

placenta the radiopaque fluid in the bladder, due to gravity, undergoes a light displacement in oblique views which is sufficient to show a change in the size of the angle of the bladder which lies higher. In marginal placenta previa (right or left) this change cannot be demonstrated as the radiopaque fluid cannot penetrate into the area occupied by the placenta, and there is no variation in the cystograms taken in oblique position.

A diagnostic sign of placenta previa, which was previously described for vertex presentation only, can apparently also be used in transverse or breech presentation: due to the pressure exerted by the fetus on the placenta previa, the upper border of the radiopaque bladder shadow shows mammillations and indentations produced by the centrally located placenta previa.—*Ernst A. Schmidt, M.D.*

Borell, U. Fernström, I., Lindblom, K., and Westman, A. The diagnostic value of arteriography of the iliac artery in gynaecology and obstetrics. *Acta radiol.*, Oct., 1952, *38*, 247–263.

Visualization of the iliac artery was accomplished by injecting the contrast medium into the femoral artery below the inguinal ligament. The needle was introduced into the artery just above a tourniquet placed on the femoral artery above the origin of the deep femoral. The opaque medium was injected in three to four seconds and the first exposure was made towards the end of the injection. Two other exposures were made two and five seconds after the first.

In all, 101 cases were studied. The following conclusions were drawn:

1. Practically all myomas cause typical changes in the appearance of the uterine blood vessels. Therefore, arteriography is of value in cases in which it is not possible to make a definite diagnosis by vaginal examination.

2. By combining arteriography with hysterosalpingography, it is sometimes possible to determine the thickness of the uterine wall.

3. The observations made on the pregnant uterus indicated that the site of the placenta can be determined by percutaneous retrograde arteriography of the iliac artery.

4. No noteworthy complications either in the arteriography itself or following the examination were observed.—*Mary Frances Vastine, M.D.*

Genitourinary System

Ellis, F. Henry, Jr., Dawe, Clyde J., and Clagett, O. Theron. Cysts of the adrenal glands. *Ann. Surg.*, Aug., 1952, *136*, 217–227.

An adrenal cyst is a rare lesion and is seldom diagnosed preoperatively. Because some of these cysts reach sufficient size to constitute diagnostic and surgical problems, the authors present an analysis of 12 such cases.

The generally accepted classification of adrenal cysts includes: (1) parasitic cysts, (2) true glandular cysts, (3) cystic adenomas, (4) serous or lymphatic cysts, and (5) pseudocysts. Because of the extreme rarity of parasitic and glandular cysts and cystic adenomas, the following classification is suggested for those usually encountered:

1. Serous cysts
2. Pseudocysts
   a. Hemorrhage and necrosis in a benign or malignant tumor.
   b. Cystic resolution of a hematoma in the absence of neoplasm.

The predominating symptoms in the cases reported were vague gastrointestinal complaints, pain, and abdominal swelling. In only 1 of the 3 pheochromocytomas was the tumor functioning. Physical findings of a mass when palpable were not characteristic.

The urographic examination was the most valuable method of diagnosis, identifying the mass as suprarenal and usually excluding a renal lesion. No further information could be obtained, however. The differential diagnosis in the cases described included cysts of the spleen, pancreas, liver, kidneys, and mesentery; retroperitoneal tumor; dermoid cyst; renal tumor; empyema of the gallbladder and aneurysm of the splenic artery.

The pathogenesis of adrenal cysts is discussed in some detail.

It is evident that, in view of the rarity of the lesion and the lack of specificity of its symptoms, a high degree of suspicion is required for the diagnosis of a cyst of the adrenal gland.—*John F. Weigen, M.D.*

Mangelsdorff, B. Die Veratmungspyelographie und ihre Verwertbarkeit in der urologischen Diagnostik. (Pyelographic demonstration of the respiratory mobility of the kidneys; its value in urological diagnosis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen ver. m. Röntgenpraxis*, Oct., 1952, *77*, 434–444.

The normal kidneys move with respiration, according to observations of the author and others, by as much as 11.8 to 32.3 mm. This was demonstrated by films taken during intravenous or retrograde pyelography, the patient being instructed to breathe during the exposure. Decreased or abolished mobility of the involved kidney was an almost constant symptom in paranephritic abscess and carbuncle of the kidney, it occurred in 75 per cent of cases of pyelotomies, in 50 per cent of stones in the kidney or ureter, and in less than half of the cases of hydronephrosis and pyelonephritis.

The decreased mobility is caused either by an inflammatory process involving the perinephritic

soft tissues or a reflex immobilization of the diaphragm, the muscles of the black and the abdominal wall; both processes may be present, the inflammation being the cause of the muscular reflex.

This paper is based on 130 pyelograms of which 30 showed decreased mobility of the kidney. The method seems to be of value mainly in early stages of paranephritic abscess.—*Karol E. Matzinger, M.D.*

Mullen, Wylie H., Jr., and Engel, William J. Circumcaval ureter. *Radiology*, Oct., 1952, *59*, 528–537.

Circumcaval ureter is a rare urinary aberration characterized by passage of the ureter behind an anomalous vena cava.

The 18th case to be correctly diagnosed preoperatively is reported and the literature is analyzed. The right ureter is always involved, with the left occasionally superadded. Hydronephrosis and hydroureter result, with a deviation of the dilated ureter in an S-shaped curve nearly to the midline. The ureter passing behind the abnormal vena cava forms an arcuate curve on pyelograms, with no filling distal to this. Nephrectomy or plastic releasing operations have been done in most cases.—*William A. Goodrich, M.D.*

Jeffcoate, T. N. A., and Roberts, Henry. Observations on stress incontinence of urine. *Am. J. Obst. & Gynec.*, Oct., 1952, *64*, 721–738.

The condition is due to an abnormal relation of the posterior urethra and bladder and is independent of bladder loading or intravesical pressure. This altered relation is demonstrable by lateral urethrocystogram only. There is alteration of the normal "T" angle of the urethrovesical junction so that the posterior right angle is obliterated and tends to form a straight line of the posterior bladder wall and urethra. Since this position obtains normally only during micturition, the authors feel that the change in the angle is the true cause of incontinence.

Iodide urethrocystography is comprehensively reviewed. Examples of stress incontinence without prolapse and in prolapse without stress incontinence are given. The posterior urethrovesical angle is obliterated in the former and not in the latter instance. Funnelling as a diagnostic sign is discounted. Several cases of incontinence due to diverticula in the trigone but without change in the urethrovesical angle are noted. The mechanism here is not clear.

Treatment has been with the Aldridge sling operation with or without anterior colporrhaphy. Unless the normal urethrovesical angle is restored, incontinence persists.

The postulated cause of most cases is in engagement of the fetal head. The posterior wall of the bladder is pushed forward, coming to lie in a straight line as regards the urethra. Stretching or injury at this time completes the process.

It is pointed out that no true sphincters are present in the urethra but that sphincteric function lies in the urethra itself. Three groups of muscles are recognized: (1) intrinsic involuntary, (2) intrinsic voluntary and (3) extrinsic or accessory voluntary. The possible interplay of fascial planes is discussed.

The authors enter a plea for complete urethrocystography preoperatively. They suggest that in some cases treatment other than surgical should be undertaken and that all results should be checked with lateral urethrocystography.—*P. B. Parsons, M.D.*

Abeshouse, Benjamin S., and Ruben, Malcolm E. Prostatic and periprostatic phlebography. *J. Urol.*, Sept., 1952, *68*, 640–646.

There is need for further knowledge of the plexus of veins which have been disclosed by the retropubic approach to the prostate and vesical neck.

The technique for preoperative examination of these vessels is to inject 70 per cent urokon percutaneously into the deep dorsal vein of the penis. This results in the demonstration of the veins of the pelvis including the upper femoral, internal and external iliac, the obturator veins and the veins of the prostatic plexus. From the roentgenograms, it appears that there are no valves in the deep pelvic veins. In the normal patient, a rich network of vessels courses over the prostate being more or less contiguous with one another, thus indicating a normal prostate gland. The typical picture of benign hypertrophy is indicated by separation of the lateral prostatic plexuses and an increase in vascularity. In carcinoma of the prostate, there is a relative paucity of veins, lack of distention and areas of irregular filling of the periprostatic veins. The demonstration of thromboses in this group of veins is suggestive of malignancy. Thrombosis of the deep femoral veins have also been demonstrated by this method.—*George W. Chamberlin, M.D.*

Fitzpatrick, Raymond J., and Orr, Louis M. Pelvioprostatic venography: preliminary report. *J. Urol.*, Sept., 1952, *68*, 647–651.

The authors report their technique for doing phelebograms of the pelvic plexus and the results in 5 cases which they have studied. The method consists of exposure of the deep dorsal vein of the penis and the injection of 10 to 15 cc. of diodrast solution with appropriate roentgenograms made during the injection. In addition to the venous injection, they use an opaque medium in the bulb of the Foley catheter in the bladder to identify the vesical neck. In 2 of the 5 cases, the venous plexus appeared to be normal, in 1 there was a paucity of vessels and lateral displacement of the plexus suggestive of carcinoma. In this instance, the patient had primary carcinoma of a seminal vesicle.

The authors conclude that it is difficult to be certain, at the present time, of the value of this procedure. They call attention to the need for a number of normal patterns and for further roentgen evidence in abnormal instances.—*George W. Chamberlin, M.D.*

Skeletal System

Jeannopoulos, Constantine L. Congenital elevation of the scapula. *J. Bone & Joint Surg.*, Oct., 1952, *34-A*, 883–892.

In this paper, a series of 35 cases of congenital elevation of the scapula are discussed. Of these cases, 21 were operated upon and a follow-up study was obtained in each case. There were 25 female and 10 male patients in the series. The deformity occurred on the right side in 16 patients and on the left in 15 and was present bilaterally in 4.

Associated anatomical lesions of the vertebral system were found in the series. An omovertebral bone was present in 11 patients and was excised at operation in 10. It appeared as a roughly trapezoid or wedge-shaped chondro-osseous structure, the narrow extremity of which was continuous with a spinous process, lamina, or transverse process of one or more of the lower cervical vertebrae. In 1 patient the omovertebral bar had no bone or cartilaginous continuity with the spine. Its apex, instead, was globular or knoblike in shape and was covered with hyaline cartilage, and it articulated with a cervical spinous process as an enarthroidial joint. The omovertebral bar was short or incomplete in 4 patients, and its free cartilaginous extremity was anchored to the scapula by means of a fibrous band. In 1 patient, the omovertebral bar formed a true joint with a small osseocartilaginous protuberance projecting from the mid-vertebral border of the scapula.

One of the patients had a malformed third rib, with an absent sternocostal component of the pectoralis major. Torticollis was also noted in 4 cases. Asymmetry of the chest was a frequent finding. Lateral curvature of the spine also occurred.

Excellent correction of the undescended scapula was obtained in all the patients at the time of operation but follow-up examination revealed complete recurrence of the deformity in 6. The cosmetic end-result was good in 7 patients and only fair in 4. Spontaneous improvement of the deformity did not occur in any of the patients who refused operation. The best results in this series were obtained in patients in whom transplantation of the scapula was performed early, particularly before the age of five years.—*Ralph S. Bromer, M.D.*

Vitale, Carmelo C. Reconstructive surgery for defects in the shaft of the ulna in children. *J. Bone & Joint Surg.*, Oct., 1952, *34-A*, 804–810.

In this paper the results of successful application of radio-ulnar fusion to large defects in the shafts of the ulna in 2 children are presented. In one the defect was congenital, in the other it was due to a large osteochondroma which required resection of the lower half of the ulna. In both cases radio-ulnar fusion was performed with good cosmetic and functional results.

Vitale points out that regardless of defects of the ulna or radius, or both, so long as the elbow and wrist-joint structures are adequate, mobile and stable, surgery can be depended upon to join the fragments into a single satisfactory forearm bone. This not only retains motion at both joints, but adds stability to the forearm. Also, when the proximal end of the ulna is joined with the distal end of the radius into one bone, longitudinal growth is renewed, so that a better looking and longer forearm results.

Roentgenograms are reproduced showing the end-results.—*Ralph S. Bromer, M.D.*

Lottes, J. Otto, Hill, Lloyd J., and Key, J. Albert. Closed reduction, plate fixation and medullary nailing of fractures of both bones of the leg; a comparative end-result study. *J. Bone & Joint Surg.*, Oct., 1952, *34-A*, 861–877.

This paper is a comparative study of the end results of three methods of treating fresh fractures of both bones of the leg. The material studied included 216 fractures of the tibia. Of these, 125 were simple and 91 were compound. The fractures occurred in 183 tibias; of these, 31 were double fractures and 1 was a triple fracture of the shaft of the bone. There were 176 patients, in 7 of whom both legs were broken. Sixty-five tibias were treated by closed reduction under local or general anesthesia and immobilization in a plaster cast, 49 were treated by plate fixation, and 102 were treated by closed reduction and fixation with a medullary nail.

The authors were impressed by the facts that most fractures of the shaft of the tibia can be nailed with relative ease and safety; that, if the leg is then protected with a toe-to-groin plaster cast, the patient may be able to bear weight on the extremity in the cast in a relatively short time; and that union may be expected to occur with the fragments in good position. Union may be expected to occur somewhat sooner than if the fractures had been treated by simple immobilization in a cast without internal fixation or by plating. Oblique fractures of the tibia near the ends of the bone are not regarded as suitable for nailing and are fixed with screws. This method, the authors believe to be satisfactory in the hands of a capable surgeon. Oblique fractures in the middle third can be nailed satisfactorily, as shown by the study of their cases. They discourage the use of plates in any except experienced hands.

The paper is illustrated with numerous reproduc-

tions of roentgenograms showing progress of the cases.—*Ralph S. Bromer, M.D.*

Frank, Howard A. Hypertrophic pulmonary osteoarthropathy simulating rheumatoid arthritis; subsidence after pneumonectomy for carcinoma. *New England J. Med.*, Aug. 21, 1952, *247*, 283–285.

A male patient, aged sixty, was admitted with a diagnosis of rheumatoid arthritis. There was an old traumatic internal cartilage derangement of the left knee. Roentgen study revealed classical changes of hypertrophic pulmonary osteoarthropathy, especially in the metacarpals, and in the radial, ulnar, tibial and femoral shafts. Chest film examination showed primary malignancy of the lung, not confirmed by endoscopy or sputum examination, but resected and confirmed histopathologically. The surgical specimen revealed an adenocarcinoma located peripherally in the right upper lobe and an undifferentiated carcinoma at the confluence of the lobes.

Three months after the right pneumonectomy, the roentgen signs of osteoarthropathy had disappeared. Six months later, the patient was in good health and the remaining lung and the bones, as well as the joints, were normal, except for changes from an old injury to the left knee.

It is stressed that hypertrophic osteoarthropathy is often mistaken for rheumatoid arthritis. Careful examination, including roentgen studies, will lead to diagnosis of occasional cases of otherwise unrecognized lung carcinoma in resectable phases. Severe forms of osteoarthropathy are usually associated with primary or secondary lung cancer.—*John W. Turner, M.D.*

Sherman, Mary S. Psoriatic arthritis; observations on the clinical, roentgenographic, and pathological changes. *J. Bone & Joint Surg.*, Oct., 1952, *34-A*, 831–852.

Discussions in case reports of psoriatic arthritis usually center around the question of whether it is to be distinguished from rheumatoid arthritis. As a rule, the consensus is that such a distinction is unwarranted. In this paper, the conclusion is reached that there is a type of arthritis which is peculiar to persons who have psoriasis and which has a predilection for the distal joints of the hands and feet. The roentgenographic changes are characteristic and are quite different from those of rheumatoid arthritis. The earliest changes are seen in the interphalangeal joints and consist of marginal erosion at the very edges of the phalanges. A little later irregular destruction is apparent, extending along the shafts of the bone from the margins so that the cortex appears "scalloped" and may contain punched-out areas. Still later gross destruction of the bone ends may be seen and they may eventually be so far worn away that they look as though they had been put through a pencil sharpener. There is usually little evidence of healing, but occasionally there is overgrowth at tendon insertions which produces cup-like deformity. There may also be irregular nodules of new bone formation near involved joints. Rarely, and much later than is the case with rheumatoid arthritis, bony ankylosis of a joint occurs.

The pathological changes in psoriatic arthritis are constant, but are not sufficiently specific so that the diagnosis can be made with certainty from the microscopic section alone. Both arthritic and skin manifestations respond to ACTH and cortisone, but the former respond to significantly lower doses. Improvement with hormone therapy was not found to be permanent. The disabling deformities of the toes can be greatly relieved by radical surgery.

The paper is profusely illustrated with reproductions of roentgenograms, pathological sections and clinical photographs of patients.—*Ralph S. Bromer, M.D.*

Stahl, S. S., Wisan, J. M., and Miller, S. C. The influence of systemic diseases on alveolar bone. *J. Am. Dent. A.*, Sept., 1952, *45*, 277–283.

Three hundred male patients ranging in age from twenty-two to sixty-nine years were divided into two groups—*Group A* consisting of 200 patients suffering from systemic diseases of at least one year's duration, and *Group B* consisting of 100 patients suffering from fractures and other local ailments of short duration.

It was found that alveolar crest resorption was more severe with increasing age, that certain disease entities, particularly those associated with disturbance of protein metabolism, were followed by more severe alveolar crest absorption than other diseases, and that the tendency to increased alveolar resorption with aging was aggravated by the presence of systemic disease. An equal degree of absorption was found in anterior and posterior teeth.—*Leslie K. Sycamore, M.D.*

Moehlig, Robert C. Osteitis deformans; familial constitutional heredity background as etiological factors *J. Michigan M. Soc.*, Aug., 1952, *51*, 1004–1007.

The author recommends the use of the term osteitis deformans, rather than Paget's disease, in order to keep from confusing it with the disease of the nipple bearing the same name. The fact that osteitis deformans is a constitutional disease with hereditary factors playing a major role is stressed and references are given in support of this concept. The close relationship between carbohydrate metabolism and osteitis deformans is fully discussed. The author had previously reported on the high incidence of familial diabetes mellitus, tallness and obesity in this disease.

He now reports a case of a father and mother having diabetes mellitus and two sisters and a brother having osteitis deformans. Both sisters had a diabetic type of glucose tolerance curve.

A further suggestion that the pituitary gland might play a leading role in the constitutional hereditary background of the disease is based partly on the fact that the pituitary gland is a factor in carbohydrate metabolism, in the development and function of the osseous system, and is also a factor in fat metabolism. This family, in which both parents had diabetes mellitus and all three siblings had osteitis deformans, led to a study of the carbohydrate metabolism and reference is made to the previous report.

The author points out that other bone diseases associated with a disturbance of the carbohydrate metabolism are acromegaly, gigantism, pituitary basophilism, thyrotoxicosis, osteoarthritis and hyperparathyroidism. These are discussed to some extent and attention is called to the important link of phosphorus and phosphatase in the carbohydrate metabolism and bone activity. He also points out that the pituitary gland influences both phosphorus and phosphatase metabolism and thus may play a definite role in some of these bone diseases. Lastly, he states that there is a reciprocal relationship between the pituitary gland and lung function. This has to do with oxygen consumption and erythrocyte formation and since the so-called chronic pulmonary osteoarthropathy is associated with impaired lung function, the latter may also be due to pituitary disturbance.—*Robert D. Moreton, M.D.*

Rosenkrantz, J. A., Wolf, Julius, and Kaicher, John, J. Paget's disease (osteitis deformans); review of one hundred eleven cases. *A.M.A. Arch., Int. Med.* Nov., 1952, 90, 610–633.

The records of 111 patients with Paget's disease of the bone are reviewed. The true incidence of Paget's disease in the adult population would be much higher if detailed skeletal surveys were to be made of the older population. In this survey the incidence was 1 per 850 patients admitted.

The exact causative factor of osteitis deformans is unknown. It is now believed that there is some diminished endocrine activity in older persons and as a result there is an increase in pituitary growth hormone which has a direct influence on osteoblastic activity and bone metabolism with consequent elevation of the serum alkaline phosphatase.

The average age of this group was fifty-five years and the average age of onset was forty-one years. Seven of the patients had relatives with Paget's disease.

A large number of patients had no symptoms referable to the disease, but when symptoms were a feature, bone pain was the predominant complaint. Defective hearing, visual disturbances, headaches and urinary disorders with calculi formation were common findings. A number of patients also developed salivary calculi and 8 had sarcomatous changes. Likewise, because of unstable calcium metabolism, calcification of the valves of the heart was observed rather frequently. In case of pressure on the cranial nerves by the increased bone production in the skull, neurological symptoms and findings may be common. Platybasia may develop, producing sensory changes and muscular weakness.

Roentgenologic bone involvement was divided into four grades. In Grade 1, the finding was that of osteoporosis circumscripta of the skull with no other manifestations. In Grades 2, 3 and 4 there was definite structural enlargement of the bone, with associated thickening of the cortex and prominence of the bony trabeculations.

It was noted that with greater bone involvement the amount of alkaline phosphatase increased, and that most of the patients who developed sarcomatous change had lesions classified as Grade 4.

The pelvic bones were most frequently affected, followed by the skull, femur and spine. In this series, 17 patients developed pathological fractures, most of which healed with callus formation.

It is pointed out that in osteitis deformans, the lesion initially is purely a destructive one. The destruction makes the bone more susceptible to stresses and strains with consequent bone production and increase of the alkaline phosphatase.

There is no specific treatment for Paget's disease. Corticotropin causes subjective improvement but has no effect on the bone lesions.—*Wallace M. Roy, M.D.*

Seghini, Giuseppe. Del trattamento delle pseudo-artrosi diafisarie delle ossa lunghe.—Revisione del nostro materiale.—Considerazione sui metodi nostri ed altrui.—Conclusioni. (On the treatment of diaphyseal pseudarthrosis of the long bones. Review of our material. Consideration of our methods and those of others. Conclusions.) *Arch. di chir. ortop. e di med.*, July, Aug., 1952, *17*, 315–349.

The author reviews 42 cases of pseudarthrosis in long bones treated in the Orthopedic Clinic of the University of Genoa and at the Institutes of St. Carona under the supervision of the directors, Dr. Raffaele Zanoli and Dr. Luigi Morasca, respectively.

To institute proper therapy for nonunion in long bones the causes should be evaluated. Among the general etiological factors are age, wasting diseases, chronic infectious diseases, endocrine disturbances, alterations of the calcium-phosphorus ratio, Paget's disease and the avitaminoses. Among the more direct causes are:

(1) Errors in reduction of the fracture
    (a) Displacement of the fragments with loss

of the stimulus to bone production by weight bearing.
(b) Delayed reduction with a cuff of fibrous callus covering the osseous ends impeding union.
(2) Errors in immobilization
(a) Inadequate, incomplete immobilization of fragments in cast.
(b) Insufficient immobilization time.
(3) Separation of fragments
(a) Excessive traction.
(b) Interposition of soft parts.
(c) Loss of bone substance.
(d) Serious local trauma to nerves, muscles and blood vessels.

In the treatment of nonunion in the femoral shaft, the author concludes that the method of choice is fixation by the intramedullary nail of Kuetschner, with autogenous bone grafting either of cancellous bone from the iliac crest or of cortical bone from the tibia.

In the treatment of pseudarthrosis in the tibia various methods of fixation were used, *i.e.*, vitallium screws, metallic plate and screws, autogenous and homogenous bone grafts.

In the treatment of the large osseous defects following chronic osteomyelitis, satisfactory results were obtained by the use of twin autogenous on-lay grafts fixed by circumferential catgut as practiced by Dr. Rinonapoli.

The problem of congenital pseudarthrosis is a difficult one from the viewpoint of both etiology and treatment.

After a review of the literature, the author favors the theory that congenital pseudarthrosis is related to a mesenchymal dysplasia with cessation of bone growth, secondary to disturbances of the nervous and endocrine systems.

Three cases of congenital pseudarthrosis are included in this survey. The first was unsuccessfully treated by autogenous fibular graft. Results in the second were poor because of resorption of a sliding graft. The third case showed firm union following the use of three homogenous tibial grafts and fixation by vitallium plates and screws.

In the treatment of nonunion of fractures of the forearm, the author concludes that the use of autogenous grafts stimulated bone growth and was more satisfactory than fixation with metal plates and screws.

In conclusion, the correction of pseudarthrosis in the long bones requires a careful estimation of the general and specific causes, open reduction, operative revision of the fractured surfaces, adequate fixation and immobilization. The use of autogenous grafts both because of the support they afford and because of the stimulation to bone growth is recommended. The indication for the intramedullary pin is principally in malunion of the shaft of the femur, humerus and ulna. The use of circumferential metal bands is condemned.—*Anthony A. Blasi, M.D.*

Moberg, Erik. Further observations on 'corticalisosteoide' or 'osteoid osteoma.' *Acta radiol.*, Oct., 1952, *38*, 279–293.

In 1941, the author reported 8 cases of a condition which he described as "corticalisosteoide." Because of interrupted communications caused by the war he did not realize until later that Jaffe had described the same bone condition as "osteoid osteoma."

Since 1941, the author has observed 4 more cases which he summarizes in this article. He notes that a large percentage of the cases from the United States are reported as having the lesion situated in the short spongy bones and often in the vertebrae while this type of bone is seldom described as being involved in the European cases.

Conclusions.

1. The disease is a distinct entity and is not inflammatory in nature.

2. The cause and character of the condition are as yet obscure.

3. Clinically the disease has a chronic course and lasts for months or years. The symptoms take the form of local pain, frequently nagging in character. It is often typically nocturnal. A palpable tumor may be present.

4. The condition has been described as occurring in all parts of the skeleton with the exception of the skull and the clavicle. In the long bones it is usually situated in the diaphyses. No more than one lesion in a given individual has been described.

5. At operation a sclerotic cortical thickening is found; at the site corresponding to the rarefaction on the roentgenogram, a central noncystic nidus of softer consistency is usually present. The size of the nidus varies from 1 mm. to the size of a bean.

6. Bacteriologically the process is sterile.

7. Microscopically the central softer area is seen to consist of osteoid tissue alternating with newly formed bone and older osseous trabeculae in irregular arrangement. The area between the eburnated bone and osteoid tissue is highly vascularized. Giant cells may be seen. There are no signs of inflammation.

8. The roentgen picture is most characteristic when the process is situated in the diaphysis. There is a fusiform tapering expansion and thickening of the cortex. A radiolucent center is usually seen in the dense cortex.

In the spongy bone the surrounding sclerotic bone reaction is much less marked or entirely absent. The central nidus in these cases is more globular and the process seems to develop in or adjacent to the cortex.

9. It may be difficult roentgenologically to differentiate the lesion from bone abscess, other inflammatory changes in the bones without abscess formation, certain forms of tumor and callus formation.—*Mary Frances Vastine, M.D.*